

THESIS  
ON  
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SPLENIC ANAEMIA AND BANTI'S DISEASE.

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## INTRODUCTION.

Greisinger and his assistant Gretscl in 1866 (Berl. Klin. Woch), were the first to employ the term "anaemia splenica" for cases of anaemia with chronic enlargement of the spleen, supposed to represent a splenic form of pseudoleukaemia.

The first systematic description of the disease was published by Professor Guido Banti of Florence, (Arch. d. Scuola d. Anat. patol Firenze 1883 11 53), who reported three cases, two with autopsies, under the title of "anaemia splenica", though he believed the disease a splenic form of pseudoleukaemia. In 1894 Banti described in the French and German Journals - Semaine Med. Paris 1894 XIV 318; and Bertrage Z. pathol. anat. U.Z. allgem. Pathol. Band XXIV, Heft 1,21 the form of the disease, characterised by progressive anaemia, splenomegaly, cirrhosis of the liver and Ascites to which subsequent writers have attached his name - Banti's disease. Banti regarded the disease as a direct result of the splenic affection and not in the usual inverse sense of the splenic enlargement being due to the cirrhosis of the liver. He considered it in all probability a toxæmia; the toxin originating from the blood itself, and not as Hollins and other writers suggest from the intestine.

Splenomegaly in association with leukaemia was well

known since its discovery by Hughes Bennett and Virchow - 1845, but a condition of splenic enlargement without any increase in the leucocytes of the blood was not previously recognised as a clinical entity.

Cohnheim however described under the term pseudo-leukaemia a condition characterised by idiopathic splenic enlargement with anaemia but no leukaemia and Gretscl in 1866 described a similar case in a child. The latter used the term splenic anaemia.

H. C. Wood, Strumpell and others published reports of similar cases under a variety of designations, such as splenic cachexia, splenic pseudoleukaemia, lymphadenoma splenica, and spleno-megalia primitiva but it was not recognised as a clinical entity until Banti's work.

In France Debove and Bruhl - Gaz. d. hop. Paris 1891. LXIV 241; Arch. gén. de, méd. Paris 1891, 1, 673 and 11 160: had described a condition of this nature under the term 'Splénomégalic primitive' a term which has since been adopted by other writers.

In 1900 and 1902 Osler in the American Journal of the Med. Sciences, Jan. 1900; and Transaction of the Assocn. of American Physicians 1902; brought the subject up to date in very thorough reviews, adding careful notes of fifteen cases of his own, a larger number than had been reported by any other writer.

Osler defines this affection as a disease, probably an intoxication of unknown nature, characterised by a course of extraordinary chronicity with no tendency to spontaneous cure; progressive enlargement of the spleen which cannot be correlated with any recognised cause; anaemia of secondary type with low colour index; Leucopenia; tendency to periodic gastro-intestinal haemorrhage and haemorrhage occasionally from other mucus surfaces; Cirrhosis of liver with ascites - Banti's disease proper - as a terminal stage in some but by no means all cases.

The term splenic anaemia has however been used rather loosely and cases of varied clinical and pathological character have been described under this heading.

I have recently had the opportunity of investigating four cases of this condition and also perusing the note of a previous case which had been in the General Hospital, Bristol, and in the present thesis it is proposed to give an analysis of the recorded literature of the subject, to attempt a differentiation of the various conditions described under the term Splenic Anaemia and to show the relationship of the Clinical condition known as Splenic Anaemia or Banti's disease to other forms of Splenic Anaemia.



ETIOLOGY.

The cause of the disease is unknown. Blood cultures, puncture of the spleen in life, cultures of splenic tissue, animal inoculations have proved negative. Examination of the organ after death has not succeeded to reveal the casual organism.

The relation of the spleen to this disease has been the centre round which there has been much discussion.

Since Banti's first description of this disease, the view most accepted now, is that the spleen is the headquarter of an extensive chronic intoxication which has its origin in the gastro-intestinal tract, conveyed to the spleen by the blood-stream. The resulting anaemia is considered by some of Banti's followers to be due to some toxic material produced in the diseased spleen; this however has never been verified, except by Sippey's experiment - injection of 2 c.c. of splenic extract from a case of splenic anaemia into two rabbits and they died in 24 hours - which is not conclusive.

Banti believed that in this affection the spleen is primarily responsible for the morbid phenomena for the following reasons:-

1st. The splenomegaly precedes the anaemia while cirrhosis of the liver appears last of all.

2nd. The pathological changes which he applies the name "fibro-adénie" are held by him as peculiar to the disease.

3rd. The curative effect of splenectomy seemed conclusively to prove that the enlarged spleen plays an important role in the causation of this disease.

4th. He believed that toxic substances elaborated in the diseased spleen causes the anaemia and later endophlebitis of splenic vein and cirrhosis of liver. Banti did not hold the view that the toxin was conveyed by the blood stream to the spleen from the gastro-intestinal tract, but he considered that the toxin or micro-organism is derived from the blood itself. The basis upon which Banti made the last statement was that whereas hepatic cirrhosis due to digestive disturbance was considered to show enlargement which passed into the atrophic stage only after many years, in Banti's disease the liver is reduced to a state so-called complete atrophy in the course of a few months.

Let me now describe the views of the various authors on the above mentioned reasons upon which Banti based his conclusions that the spleen was primarily responsible for the morbid phenomena; and then to conclude the etiology with the various opinions that are held as to the cause of this affection.

Ledingham in Allbutt's System of Medicine, says:-

"The early appearance of the splenomegaly, however has been attested by numerous observers, and this has been a stumbling block to those critics who maintain that a cirrhosis of the liver is the cause both of the splenomegaly and the anaemia or that some toxic agent affects the liver and spleen simultaneously. Now there is no doubt that the splenomegaly which occurs in 50-80% of all cases (Klopstock) of cirrhosis of liver is not due simply to stasis in the portal circulation, as it appears before stasis is established. Further the histology of the spleen in hepatic cirrhosis differs considerably from that found in simple stasis. In cirrhosis there is, as a rule hyperplasia of the pulp, but in some cases there may be very considerable fibrosis of the connective tissue framework of the organ. Very large spleen such as in Banti are rare in portal cirrhosis of liver, but it may quite reasonably be supposed that in certain circumstances the unknown toxic agent responsible for the hepatic changes may at the same time induce a greater reaction on the part of the spleen than usually occurs." The pathological changes which Banti gives the name "fibro-adenie" and held by him as peculiar to the disease, Ledingham says that subsequent writers have described similar changes without however, holding that these changes were, so to speak pathognomonic and might not occur as the result of a number of morbid processes. The lesions may indeed be fairly described as

those common to the Leucopenic splenomegalies of non-parasitic origin. Marchand found the same changes in a case which was in all probability of Syphilitic nature and put forward the view that many cases of so called Banti's disease may be due to permanence of congenital syphilitic splenomegaly.

Banti and his followers maintain that as splenectomy is curative, it serves as a strong argument in favour of the view that the spleen is primarily at fault, but as Hollins, Ledingham, and others point out this does not necessarily follow, for they suggested that the improvement may result from (1) Removal of a diseased and an excessively heavy spleen which has been causing great mechanical inconvenience or (2) setting up of an artificial collateral circulation. Moreover the subsequent history of cases in which the spleen has been removed is not sufficiently definite or of long enough duration to allow us to say that splenectomy is curative.

As to the relation of the spleen in the production of the anaemia three main theories have been advanced. (1) Spleen may give rise to anaemia by increased haemolytic activity; this hypothesis naturally suggested by enlargement of an organ which normally plays some part in the destruction of red blood cells. In 1902 Lancet, Aug. 23rd - Barr suggested that vasomotor paralysis of the splanchnic area,



caused by disease of the sympathetic leads to engorgement of the abdominal viscera especially of the spleen and liver, thus causing increased haemolysis resulting in splenic Anaemia. In 1911 - Roy. Soc. Med. - Sutherland and Burghard advocated the view that splenic anaemia is due to loss of vaso-motor control in the splenic artery leading to congestion and hyperplasia of the spleen, a condition which they compared with that of the thyroid in Exophthalmic Goitre. Hollins, Rolleston and others objected to the above theory on the ground that the enlargement of the spleen in splenic anaemia is not a pure hyperplasia but is due to a chronic fibrosis. This being so diminution rather than increase of function should be expected. Further as Rolleston points out the blood picture is different from those of pernicious anaemia or of haemolytic jaundice - diseases in which blood destruction is known to occur and there is also no pigmentation of spleen or liver to support the view of increased haemolysis in the spleen.

(2) The spleen may be the headquarters of a chronic infective or toxic process, conveyed by the blood stream and possibly derived from the alimentary tract. The resulting poison may cause the local splenic changes and later inhibit the blood formation, both erythroblastic and leucoblastic, so producing anaemia. This theory has the great merits of explaining satisfactorily the subsequent appearance of the hepatic cirrhosis. Against this theory

are (1) There is no convincing evidence that the anaemia is due to inhibition of blood formation, for after a haemorrhage the blood condition may steadily improve until another bleeding occurs. (2) It is extremely doubtful if the spleen has a haemopoietic function in adult. (3) No toxin have been isolated from the spleen.

(3) Enlargement of the spleen due to some unknown cause, may induce anaemia as the result of haemorrhage, which is brought about mechanically. Thus torsion of the splenic vein would divert the venous blood from the spleen into the vasa brevia, the normal blood current being reversed, and caused great dilatation of these veins and so oesophageal varices from which copious or occult bleeding might result. This theory satisfactorily explains the anaemia but not the subsequent hepatic cirrhosis of Banti's disease.

The various views which are held as to the cause of this affection.

1st Infective Origin; (a) Hollins (Practitioner XCIV p. 459) suggests that this affection is due to active haemic intoxication and that the colon bacillus can cause all the features of this disease. He believes the hypertrophy and fibrosis of the spleen are due to active intoxication by the colon bacillus and the anaemia due to a specific haemolysis. The way in which the colon bacillus act is by producing colilysin which destroys the red blood cells. Hollins holds that the similiarity

between the clinical manifestation of Banti's disease and cirrhosis hepatitis together with the fact that in cirrhosis enlargement of the spleen is present in 50-80% of cases (Klopstock) suggests that both affection have a common bacillaemic origin. The finding of Adami of polymorphic forms of bacillus coli in cases of cirrhotic liver is held by Hollins to strengthen his view that bacillus coli is the cause of both affections. Hollins was led to form his bacillus coli theory from the study of his three cases and also from the experiments he carried out by inoculations of rabbits with bacillus coli. From the three cases certain clinical facts made him conclude that the affection is due to poisons absorbed from the gastro-intestinal tract and that these are probably due to bacillus coli. The clinical facts were:-

- (a) Two female patients had history of constipation.
- (b) Oral sepsis was present in one case.
- (c) Enormous number of bacilli coli found in diarrhoeic motion preceding death.
- (d) In one case nervous lesions resembling those artificially produced by injection of animals with bacillus coli.
- (e) The evidence of other diseases associated with invasion of bacillus coli especially coliuria.

(f) The presence of bacillus coli in the normal spleen.

(g) Cirrhosis of liver is produced by toxin from gastrointestinal tract and probably by the bacillus coli.

(h) The form of anaemia is chlorotic and Dr. Andrew Clark is emphatic in stating that constipation, which is really a copraemia due to absorption of poisons from the large bowel is the cause of chlorosis. The nature of the poisons has not been definitely determined, but Hollins thinks it is reasonable to conclude that one of these poisons may be a specific toxin from the bacillus coli.

The results of his experiments showed that rabbits inoculated with bacillus coli showed that their red blood cells were decreased from 6,304,000 to 3,840,000 therefore a reduction of 45% and an increase in the weight of the spleen; and lastly the product of spleen of an inoculated rabbit had a haemolytic action on the red blood cells of a normal rabbit. This haemolysis being due, according to Hollins, to a toxin known as Colilysin.

(b) A. G. Gibson - Lancet, Nov. 1st 1913 - holds the view that some cases but not all of Banti's disease are due to a streptothrix invasion for the following reasons. Firstly Banti's disease is cured by splenectomy; Secondly it reacts satisfactorily to treatment by salvarsan and bore



a resemblance to syphilitic cirrhosis as Sir Wm. Osler had pointed out in one of his cases - Congenital syphilis of Liver with the Picture of Banti's disease. Lastly he exhibited the spleen of a fatal case, the organ showed generalised fibrosis, the surface was greyish pink and dotted throughout it were small areas of pigment. Microscopically these areas showed about the splenic veins masses of black staining material, which consisted of segmenting threads, which on careful examination were without doubt to be growth of streptothrix. He found these growths also in three other examples in the Radcliffe Infirmary, and also in the spleen and cavity of a tuberculous case, but he has failed to obtain a culture of these organisms. These parasites were found in five out of 100 cases of spleen examined.

(c) Owen Richards - American Journal Medicine Sci. 1913 11 863 - contributed an interesting paper upon the curative effect of splenectomy on a peculiar malady known as Egyptian Splenomegaly. The main features of this affection are enormous enlargement of spleen, the average weight being 3 lbs; moderate enlargement of the liver; slowly progressive <sup>an</sup>asthenia and ultimate death preceded by ascites and he went on to say that to some extent this malady resembles Banti's disease but it differs from the latter in that it is associated with fever and that the hepatic cirrhosis instead of being a terminal stage of

splenic anaemia as in Banti's disease is an essential part of Egyptian Splenomegaly from its commencement. He regards this disease as probably due to an infection as yet unknown.

2nd Traumatic.

Ledingham - Allbutt's system of Medicine - lays stress on traumatism as a possibility factor in the etiology of Splenic Anaemia and Banti's disease. He points out that several cases, (Carr, Armstrong, Barr, Harris and Herzog, and Fichtner), showing the symptoms of Splenic Anaemia or Banti's disease have presented a history of severe abdominal injuries and that thrombosis of the portal vein with consequent splenomegaly, anaemia and moderate cirrhosis of the liver has been proved on several occasions to follow injury (Heller, Ponfick, Saxer and Schmorl). He says "a swelling of the spleen has indeed been noted within a few weeks of the occurrence of an abdominal injury. Several cases also have been found to date their illness from a protracted and difficult pregnancy and Enteric fever has also figured in the history of one or two cases". Ledingham thinks that the thrombosis may be the result of an earlier pylethrombosis.

(3) Obstruction of Portal System - Watkins - Internat. Clinics Vol. IV 20th series 1910 - after bringing forward a mass of pathological evidence from recorded cases and cases which he has personally seen, says "it is clear

that in main the complex of splenic anaemia and Banti's disease, as formulated by Osler, is in certain cases if not in all, the result of an obstruction to the splenic and portal circulation, particularly the former, and that this obstruction is most commonly the result of an old thrombophlebitis of the portal and its radicles. Until it has been definitely shown that splenic anaemia or Banti's disease can exist without evidence of obstruction of the portal or splenic vein, then my conclusion must hold that the complex is no disease entity, but only as symptom and pathological complex due to various causes."

Mr. Connell showed Hollins a case in Sheffield in which there was no change in the splenic or portal vein.

PATHOLOGY.

The spleen retains its usual form and is firm in consistence but is enormously enlarged, weighing usually from two to five pounds, but greater weights have been recorded. The spleen does not attain the size reached in splenomedullary leukaemia or in Gaucher's disease. The capsule is thickened, its surface is smooth but may show patches of perisplenitis and adhesions to neighbouring organs are rare; if they occur they are usually to the diaphragm.

On section the splenic substance presents a dark red appearance and a general hyperplastic fibrosis; numerous whitish points and streaks due to the fibrosed Malphighian corpuscles and trabeculae; haemorrhages and infarction have also been noted.

Microscopically - According to Banti whose description Ledingham follows. The spleen shows prominently great thickening of the trabeculae passing from the capsule to the interior. Some of the Malphighian bodies appear quite healthy but the great majority show hyaline & sclerotic changes proceeding from the sheath of the follicular artery and have on this account lost their normal appearance. Sometimes these nodes may be completely converted into fibrous tissue but it is always possible to demonstrate that the fibrosis extends from the periarterial sheaths. Banti has also noted that irregularly shaped hyaline clumps, staining deeply with Eosin present in many Malphighian bodies. In



such cases there are generally in the neighbourhood of these masses epithelioid cells undergoing a hyaline necrosis. The follicular artery may contain a hyaline thrombus.

The reticulum of the pulp shows great thickening and hyaline degeneration of its fibres, a condition which Banti called "fibro-adénie" and he suggested that it may be due to a secretion from the degenerated cells of the pulp. The venous sinuses are not dilated but are richly lined with endothelial cells, many of which lie in the lumen. Mitosis in these endothelial cells was not observed by Banti.

Dr. R. T. Williamson observed a number of phagocytic cells containing red blood cells but these have not been subsequently observed in the spleen in splenic Anaemia. Dock & Watkin found nucleated reds and Megakaryocytes in the spleens of two cases. Armstrong observed in his case numerous phagocytic endothelial cells containing blood pigments while Nager and Baumlin reported eosinophil cells in large number and pulp haemorrhages were numerous.

Hollins could not find evidence of increased haemolysis such as broken down blood cells or areas of pigmentation.

Sclerosis and calcification of the vessels of the pulp were observed in one case. The splenic vein is enlarged and tortuous, this is what would be expected as a natural result of the increased size of the organ and its liability to drag

on its pedicle. Banti observed that the portal vein and its tributaries were dilated during the ascitic stage but not in the transitional stage. Atheromatous and sclerotic plaques were generally present in the splenic and portal veins during the transitional period, and the other branches, such as the mesenteric veins, became similarly affected later. Dock and Watkins, Edens, Nancrede and others observed stenosis and calcification of the portal, splenic and mesenteric veins. Ledingham suggests that thrombosis in these vessels may furnish an important clue to the etiology of this disease. The vasa brevia in the gastro-splenic omentum are dilated, this being the result of the reversed blood current brought about by torsion of the pedicle. The veins at the lower end of the oesophagus are also frequently varicosed and may be ulcerated.

Liver. Banti states that during the transitional stage the size of the liver may be normal, but that in the ascitic stage a great diminution in its size occurs. The surface of the liver is granular and macroscopically at least, it has all the features of portal cirrhosis (Laennec), microscopically, however, the cirrhotic process is never so extensive as in alcoholic cases. Changes in the liver are not confined to those cases presenting the complete Banti syndrome. Even in splenic anaemia without any macroscopical evidence

of cirrhosis, Banti admits that a slight hyperplasia of the interlobular connective tissue occurs. Indeed from subsequent work that the condition of the liver will not enable us to differentiate between splenic anaemia and Banti's disease.

Warthin and Chiari showed that cases presenting the symptoms of splenic anaemia during life, may at the post mortem be characterised by decided cirrhotic changes in the liver, thus forming a transitional stage to the Laennec type of portal cirrhosis postulated by Banti in the final stage of the disease described by him. There is no doubt that the condition of the liver is the most variable factor in the syndrom. Osler regards that the slight enlargement of the liver in the ordinary case of splenic anaemia may be due to chronic passive congestion.

Lymphatic Glands. Banti found no enlargement of these and they appear quite normal in his cases. He alleged that in some reported cases of splenic anaemia, the lymphatic glands were hypertrophied, a condition which was never found in the Banti syndrom. Subsequent writers found that the lymph glands never shewed hypertrophy in splenic anaemia or Banti's disease.

Dock, Warthin and Stengel described atrophy of mesenteric glands with areas of hyaline and calcareous changes. These authors found a notable hyperplasia of the haemolymph glands lying along the thoracic aorta and in the

retroperitoneal region. Enormous number of cells containing red blood cells and blood pigment were present in these glands, and in view of the fibrosis which involved the loss of the haemolytic function of the spleen, they concluded that this hyperplasia of the haemolymph nodes was compensatory.

Bone Marrow. Little attention has been paid to the condition of the bone marrow in cases of splenic anaemia & Banti's disease. Banti noted that the marrow presented foetal character and that nucleated red blood cells were not numerous.

Osler says that the marrow may show the compensatory changes secondary to severe anaemia - e.g. proliferation of the erythroblastic tissue. Dr. R. T. Williamson reported large number of cells containing red corpuscles similar to those present in the spleen of his case. Dock and Warthin reported lymphoid changes, nucleated red blood cells were increased and phagocytes containing blood pigment were present. Stengel found the marrow was lymphoid in type but with absence of normoblasts.

Gastro-intestinal Tract.

In spite of the frequency of gastro-intestinal disturbances in the course of splenic anaemia and Banti's disease, the absence of severe pathological lesions affecting



the stomach and intestine is remarkable. Banti noted thickening of the intestinal wall and opacity of the peritoneal coat, but in the third or ascitic stage only.

Hedemus attaches great importance from the pathological point of view, to the presence in his case of a localised thickening of the intestinal wall at the ileo-caecal junction; the ileo-caecal valve was considerably narrowed, and the mesentry at this point was much thickened, the vessels being embedded in firm fibrous tissue, the subserous and muscular layers contained extensive areas of cellular infiltration in the neighbourhood of the vessels. The intestinal mucosa has generally been reported as atrophic. Considerable importance must be attached to the frequent occurrence of oesophageal and haemorrhoidal varices. Rupture of a cardiac varix has not infrequently been the immediate cause of a fatal issue.

Pancreas. Banti found in one of his cases, atrophy with hyperaemic spots in the parenchyma.

Dock and Watkin observed dilatation of pancreatic veins and thickening of their walls.

Stengel and Stanley reported a moderate degree of sclerosis of the organ.

Simonds noted the presence of phleboliths.

Kidneys. Little information of these have been noted. They have generally been found to show a moderate degree of

fibrosis. Hyaline and calcareous changes in the glomeruli were noted by Dock and Watkins.

Suprarenal. Stanley noted fatty change in one case and an extreme atrophy in another.

Osler regards that the condition of the suprarenals must be looked upon with some importance due to the peculiar brownish pigmentation of the skin observed in many cases.

Nervous System.

Banti recognised fundamental and striking changes in the sympathetic nervous system. He found not only changes in the semilunar ganglia and solar plexus of the abdomen but also in the ganglia of the neck. The changes consisted in lymphoid infiltration of the stroma of the ganglia, fatty and pigment degeneration of the ganglion cells and a degeneration of nerve fibres springing from the ganglia. Barr says that Banti's three cases showed changes in the circulation pointing to paretic condition of the whole splanchnic area and he looks upon this as an important determining factor in the causation of the disease.

Pathology of the Spleens, and Splenic veins and arteries under my observation.

Three of the four cases had splenectomy performed.

Case No. 1. Spleen weighs 4 lbs. 5 oz. is enormously enlarged, retains its usual form, is firm in consistence. No

haemorrhage or infarction present. Capsule is greatly thickened and show slight signs of perisplenitis but no adhesions found at the operation.

On section the splenic substance presents a dark red appearance, and a general hyperplastic fibrosis. The Malphigian bodies were difficult to see.

Microscopically. Section shows great thickening of the capsule and thickened trabeculae passing from the capsule to the interior. The reticulum of the pulp presents great thickening of its fibres, but no hyaline degeneration can be detected. Most of the Malphigian bodies show some sclerotic changes, but retain their appearance. The venous sinuses are not dilated but there is proliferation of endothelial lining cells, some of which lie in the lumen. No mitotic changes can be detected in these cells. No phagocytic cells containing red blood cells can be seen. Nucleated reds and pigments are noticed in sections. Some of the pigment is contained in some of the cells. Normal red cells, some of which are disintegrated, are present. The arteries are thickened and some contain transparent looking thrombus.

The splenic artery and veins are tortuous and elongated. On section and microscopically slight atheroma is seen.

Case No. 2. Spleen is enlarged, weighing 1 lb. 6 oz, retains its usual contour and firm in consistence. No haemorrhages or infarction can be seen. Capsule is greatly thickened and slight perisplenitis present but no adhesions.

On section the splenic substance presents a dark red appearance and the malphigian bodies are indistinct.

Microscopically. Section shows general fibrosis. The capsule is greatly thickened with thickened trabeculae passing to the interior. The fibres of the reticulum are thickened.

Most of the malphigian bodies are atrophied & sclerosed.

The venous sinuses are not dilated but have a rich endothelial lining. No mitotic change can be seen. No phagocytic cells containing red cells can be seen.

Nucleated red cells can be seen. The arteries are thickened. Thrombosis is present.

The spleen of Case No. 3. weighs 2 lbs. 3 ozs, and shows thickening of capsule; slight perisplenitis and retains its usual appearance. The average weight of the three spleens is 2 lbs. 10 ozs. and the two spleens which have been completely examined showed the same common changes namely chronic generalised fibrosis.



In summarising the various theories on the etiology of Banti's disease and splenic Anaemia, the one point which most writers agree is that the disease is a toxæmia but as to the origin of the toxin there is much discussion. Banti regarded the spleen as primarily responsible and toxic substances are produced in the diseased organ which bring about the anaemia, this however has not been verified except by the experiments of Sippey which are not conclusive, and moreover no toxin have been isolated from the spleen. The view, that the toxin inhibit blood formation, both erythroblastic and leucoblastic so producing anaemia, is doubtful from the fact, that after a hæmorrhage the blood condition may steadily improve until another bleeding occurs, and secondly it is extremely doubtful if the spleen has a hæmopoietic function in adult. Banti did not hold the view that the toxin originates in the alimentary tract, but considered that it is derived from the blood. The reason being that hepatic cirrhosis due to digestive disturbances passes into the atrophic stage after many years while in Banti's disease the so-called complete atrophy is produced within a few months. Moreover as the liver is one of the main viscera connected with the digestive tract, one would expect the liver to be affected before the spleen, which is contrary to the complex-syndrome of Banti's disease. Hollins

theory that the bacillus coli may be the causative agent is weakened by the above reasons and also by the fact that bacillus coli affection is common in the terminal stages of various affections. Hollins regards the colilysin produced by the bacillus coli haemolysis the blood, but he found no evidence of haemolysis in his cases.

Against the streptothrix theory of A. G. Gibson, is the fact that no growth of the organism could be obtained in culture and secondly they were present in five out of one hundred cases of spleen examined.

With regard to Ledingham's Traumatic theory with thrombosis of the portal vein, and Watkin's Obstruction of Portal system theory, the fact that one of the important causes of thrombosis and thrombophlebitis is toxin circulating in the blood, would make one expect to find these changes in some if not in all of splenic anaemia or Banti's diseases.

The pathological changes in the spleen, liver, pancreas, & kidneys as described by all the writers, are of a fibrotic character, while Dock, Warthin and Stengel found a notable hyperplasia of the haemolymph glands lying along the thoracic aorta and in the retroperitoneal region. In these glands enormous number of cells containing red blood cells and blood pigment were found. These changes they regarded as compensatory for the loss of the haemolytic function of

the fibrotic spleen.

The description of the changes in the bone-marrow are varied. Banti noted that the marrow presented foetal character and that nucleated red blood cells were not numerous; Osler says the marrow may show the compensatory changes secondary to severe anaemia, e.g., proliferation of the erythroblastic tissue. Dock and Warthin reported lymphoid changes, nucleated red blood cells were increased and phagocytes containing blood pigment were present. Stengel reported lymphoid change but with absence of normoblasts.

These pathological changes and the blood pictures of the cases under observation, the latter showing great intermittency in the red cell and white cell counts and the persistent presence of abnormal cells, - nucleated reds & myelocytes before & after operation; suggest that the primary lesion is a disturbance of the erythroblastic and leucoblastic function of the marrow thus producing the variability in the count of the red & white cells. This disturbance may produce a byproduct, which is a toxin producing thereby the chronic toxaemia.

The arteries and veins being the channels for the conveyance of blood and the spleen the filtering organ for the blood would naturally become affected early. The enlarged and fibrotic spleen, by its weight would further

aggravate the already existing anaemia by digestive and general bodily disturbance mechanically.

Incidence.

It is not a rare disease.

A family incidence has been noted by several writers (Brill, Bovaird, Collier, Wilson and others), although this seems to be unusual.

With regards to age, statistics vary, thus 43.3 was the average age in L. B. Wilson's collection; 15 was the average age of the sixteen British cases. Of the cases which have been recorded since 1912 the average age is 21.7.

It is a disease of young and middle life, the majority of cases occurring between the tenth and the fortieth year.

With regards to sex, statistics again vary thus out of thirty-three American cases, twenty-five were males and eight were females, but out of sixteen British cases, five were males and eleven were females, (Rolleston, Practitioner Vol. 92 p. 470). According to Banti out of fifty cases, thirty-two were females and eighteen males. Out of the sixteen cases which were collected as far as possible since 1912 the sex was mentioned in thirteen cases and seven of these were women.



### Symptomatology and Diagnosis Treatment.

The cardinal symptoms of Splenic Anaemia and its later stage Banti's disease are (1) Chronicity with no tendency to spontaneous cure. (2) Progressive enlargement of the spleen which cannot be correlated with any recognised cause, such as leukaemia, syphilis, malaria etc.

(3) Absence of enlargement of lymphatic glands. (4) Anaemia of secondary type with low colour index. (5) Absence of Leucocytosis and usually the presence of Leucopenia.

(6) Liability to copious gastro-intestinal haemorrhage, from time to time. Occasionally haemorrhage from other mucous surfaces may occur.

(7) Cirrhosis of Liver and ascites as a terminal stage in some but by no means all cases.

Professor Banti described three stages.

(1) Preascitic stage in which splenic enlargement is present with or without anaemia.

(2) Transitional stage of which the most prominent symptom is diarrhoea. Anaemia and blood changes are present; the liver shows some enlargement and jaundice present.

(3) Ascitic stage or Banti proper.

This division seems arbitrary and the dividing line seems too undefined, especially between the preascitic and transitional stage, for these stages run into one another. Chronicity is a remarkable feature. The case usually lasting for four to ten years. Osler and Irving Lyon lay stress on the chronicity while F. Taylor and Samuel West emphasize the fact that death usually occurs in six months to four years. Sutherland and Burghard say "As a matter of fact there would appear to be acute, subacute and chronic forms of the disease. It may be that the juvenile type tends to be acute as so often happens in other affection".

The duration of the cases under observation are as follows:-

Case No.1. First noticed shortness of breath in 1913 and splenectomy performed in 1916. In 1903 he had a severe blow in the abdomen and passed blood per rectum for 2 days. He was kept in bed for two weeks and was quite well again. For two or three years after this he used to suffer from 'colic' but did not notice his abdomen to be distended. Spleen was accidentally found to be enlarged whilst he was in hospital for diarrhoea in 1915.

Case No.2. First noticed shortness of breath in 1915; splenectomy performed in 1916. In 1912 patient was treated for rheumatism, her complaint then was aching

(3)

pains in the limbs. The joints were not swollen or red. The pains did not shift from joint to joint. No sweating was present. Did not improve under treatment. In 1916 was admitted for gnawing pain in the abdomen and the spleen was found to be enlarged.

Case No.3. Jaundice was first noticed in July 1913, shortness of breath in November 1913. Spleen was found to be enlarged accidentally by the doctor in 1914 and Splenectomy was performed in 1917. Patient has been treated for some years for Neuresthenia, the complaint then was aching in the limbs.

As a rule the case is well developed before being seen. The symptoms are of gradual onset and the first complaint may present:-

(1) Those which result from Anaemia such as loss of strength; extreme debility; tired feelings; shortness of breath; palpitation; irregular or cessation of menses; and in more severe cases oedema of the feet.

(2) Those which result from the splenic enlargement such as feeling of weight; distension or dragging in left side of abdomen with sometimes the addition of pain, vomiting, diarrhoea or constipation.

(3) Haemorrhages from mucous membrane; of these epistaxis is perhaps the commonest and earliest met with. Next in frequency is haemorrhage from the alimentary tract,

haematemesis or melaena or both. Gastric haemorrhage may be severe and frequently repeated and is sometimes fatal. Of more rare occurrence is haemoptysis, oozing from the gums, haematuria or menorrhagia. In severe cases petechial haemorrhage into the skin can be seen.

Lastly jaundice or Ascites may be the first complaint.

Splenomegaly. The enlargement of the spleen probably always precedes, sometimes for long periods, the anaemia; and the condition if discovered in this early stage, appears to be one of simple splenomegaly.

The enlargement which is progressive varies according to the duration of the disease. It may be very large but, as a rule, it rarely attains the size of the spleen in spleno-medullary leukaemia or in Gaucher's disease. It usually reaches the navel and often the anterior superior spine of the ilium, and it may even pass the median line and occupy a considerable part of the right side of the abdomen. The enlarged organ feels smooth, firm and retains its characteristic shape with its sharp edge and notches presenting on the anterior margin. Tenderness on pressure, a feeling of discomfort or of weight or fulness may be present, but pain is rarely present unless perisplenitis or infarction occurs. The organ moves with res-



piration unless adhesions are present.

Case No.1. Noticed shortness of breath first in 1913. The splenomegaly was found accidentally during examination whilst patient was in hospital in Australia in 1915.

When examined on admittance to the General Hospital on 27th March 1916 the spleen was found to extend downwards 1" above the anterior superior spine and the anterior border was well beyond the umbilicus - as shewn in the diagram. The spleen was firm, smooth and retained its characteristic shape, with its sharp anterior border and the notch. No tenderness or pain but feeling of weight is present. Under weekly X-ray treatment the spleen varied in size slightly and on the whole after about six months' treatment it remained practically the same size as when seen on admittance. No murmur or rub can be heard on auscultating over the enlarged organ. The liver was not enlarged. No ascites was found.

Case No.2. Noticed shortness of breath in 1915. The spleen was first noticed to be enlarged when patient was examined on admission to the General Hospital, Bristol on 14th August 1916.

The lower pole of the spleen was found to extend to about an inch from the umbilicus and in the mammary

line the lower border extended 4" from the costal margin. Its characteristic shape was retained and the notch could be felt distinctly in the sharp anterior border. The surface was smooth and the consistence was firm. The organ was tender and patient complained of pain in the upper left side of abdomen. A bruit was heard over the spleen.

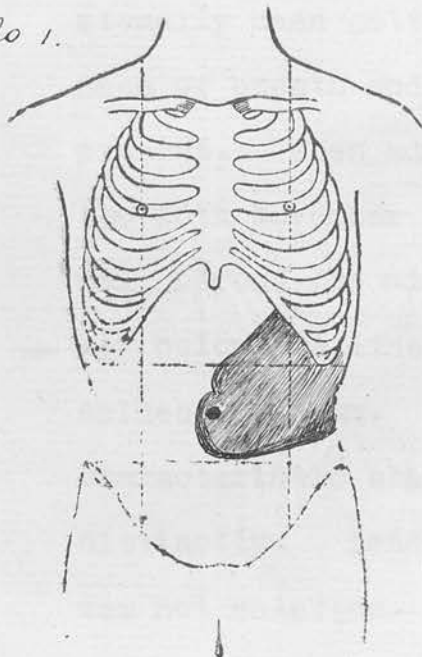
X-ray treatment was resorted to but with no improvement. The liver was slightly enlarged and no ascites found.

Case No.3. Noticed jaundice in July 1913; shortness of breath in November 1913 and spleen was accidentally found to be enlarged by the doctor in 1914. On admission on May 29th 1916 the anterior border of the spleen extended to the umbilicus, in the middle line of the abdomen the spleen extended to the level of the anterior superior spines. The notch was felt. The organ was firm and smooth. Pain in upper part of abdomen was present. Liver was enlarged; the lower border being 1" below costal margin in the parasternal line. Shifting dulness was also noticed in the abdomen - Vide diagram.

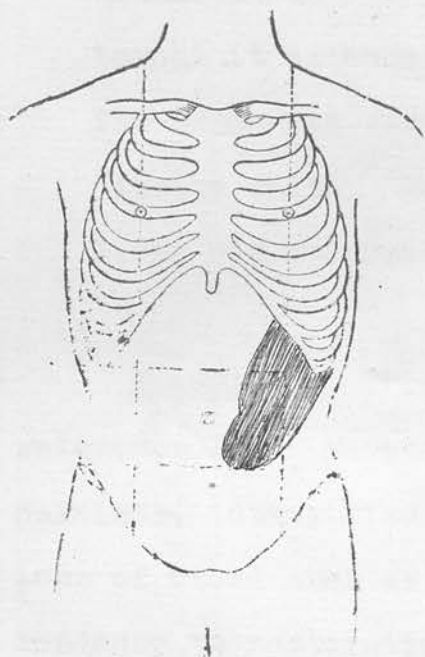
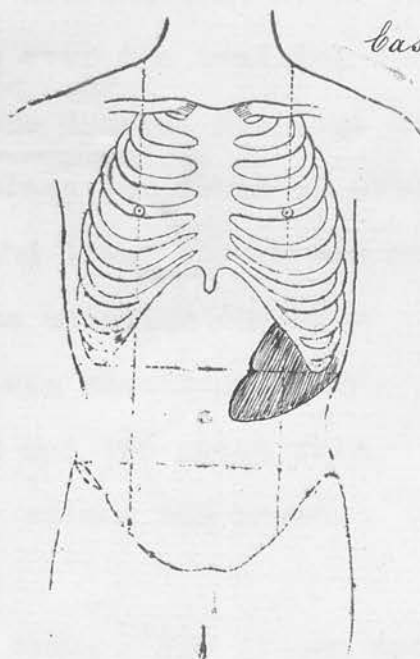
Case No.4. Noticed a hard lump in the left side of the upper part of the abdomen in 1915. This swelling could be moved about easily. Since then it has

Diagrams of Spleen  
on  
Clinical Examination

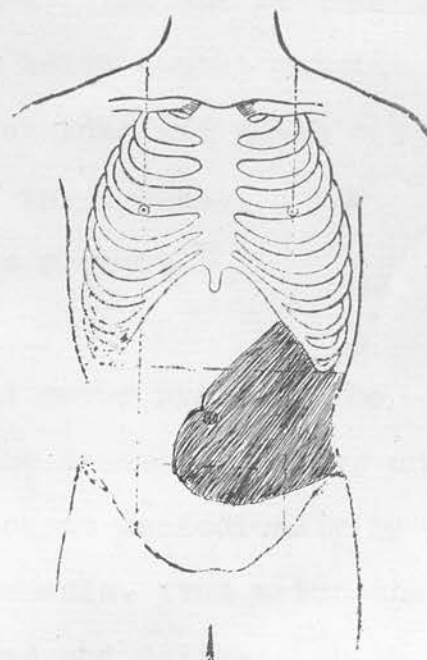
Case No 1.



Case No 2



Case No. 3



Case No 4

steadily been getting larger, and sometime after shortness of breath and tenderness over the swelling were present. When admitted to the General Hospital on the 30th November 1915 the spleen was found to extend well beyond the middle line and lower border was about  $1\frac{1}{2}$ " below the line joining the anterior superior spines of ilium. The organ was smooth and firm. The characteristic shape retained and the notch felt distinctly. Tenderness over spleen was present. Liver was not enlarged.

Case No.5. Notes only have been seen. The spleen was described as very much enlarged. In the left parasternal it extended six inches below costal margin. The organ was firm; shape maintained and notch distinctly felt. No tenderness was present. The liver was enlarged and ascites present.

Anaemia. This follows but never precedes the splenomegaly. Sooner or later the anaemia develops and persists, intensified in certain cases periodically by loss of blood such as from haematemesis, from which the tendency to restoration is impaired and delayed; while in other cases without any history of haemorrhages. In the latter event Rolleston suggests that although no obvious haemorrhage occurs yet there may have been



bleeding, such as from oesophageal varices in small amount that it is not obvious to the naked eye. Hollins regards that this may be due to some toxic factor.

After bleeding has occurred, the condition of the blood usually improves and may become normal, in some instances indeed, the number of red blood cells may be more than 5,000,000 per c. mm. Rolleston reported a case illustrating and confirming his last statement. A patient in St. George's Hospital whose red cell count went up from 3,200,000 to 6,000,000 before the fatal haematemesis.

The blood changes may be summed up as follows:-

There is a reduction of red blood corpuscles.

Greater reduction of haemoglobin resulting in a low colour index and producing a chlorotic type anaemia.

Diminution of white blood cells resulting in Leucopenia.

A relative lymphocytosis.

The reduction of the red blood cells is not extreme and usually the count amounts to between three and four millions. It is rare to fall below two millions. In some cases the number of red blood cells at time of observation, is normal. In Osler's series of cases the average count is 3,425,000 with extremes of 2,187,000 and 5,200,000 per c. mm: Gilbert Barling in the Lancet 30th Jan. Vol 1. 1915 recorded a case of a patient age 19 who after three

or more days of haemorrhage from gastro-intestinal tract showed a count of 1800,000 red cells, whites 10,000 and haemoglobin 20%. And another case age 20 with no recorded history of haemorrhage showed the following count - Red cells 1,010,000 per c. mm. Whites 2,200 Hb. 50%.

In all the five cases under observation the red blood cell's count amount to between three and four millions.

According to Osler and Ledingham poikilocytosis and polychromatophilia are exceptional and only occurs when the oligocythaemia is extreme. Nucleated red blood corpuscles are not often met with and when they do occur in severe cases, normoblasts always predominate over megaloblasts. Osler says normoblasts are seen only occasionally in the advanced stages of the disease. Hollins could not see any abnormal red cells in his cases.

The reduction of haemoglobin is proportionately greater than the red blood cells so that the colour index is below normal.

According to Ledingham the reduction is not usually so great as in chlorosis & in the later stages when the number of red cells is greatly reduced the colour index may rise to normal or above it. The average reduction in Osler's series of cases is 47% with extremes of 25% & 75%.

As a rule the white cells are reduced in number. The count varies a good deal usually between 2,000 and 4,000. Osler's average was 3,850. Gulland & Goodall mention an extreme case of 800. Leucocytosis, according to most authors,

is rare except as a result of a passing complication and sometimes after a profuse haemorrhage. The differential count is inconstant. According to Ledingham if the leucopenia is considerable, the reduction seems to affect mostly the polynuclear cells so that a relative lymphocytosis results. In some cases the presence of as many as 5% of mast cells has been described and in advanced cases a small number of myelocytes may be met with.

Dr. Cowan recorded a number of atypical cells of the lymphocyte class in his two cases.

In post operative cases Dr. Cuthbert Ede, quoted by Sir Clifford Allbutt and his colleagues in the British Medical Journal, March 11th, 1916, formulated the following conclusions:-

The first and best marked change in the peripheral blood is an increase in the total number of the polymorphonuclear cells, and a large increase in the number of red cells. This is instantaneous.

(2) During the first year after the operation there is an increase in the absolute number of the lymphocytes. Later these become normal in number, but eosinophiles are increased.

(3) Finally the blood picture resumes a perfectly normal state.

Noguchi, quoted by Dr. Ede, comes to the same conclusions, and they are confirmed by Captain Jones' case in

the British Medical Journal February 5th 1916, and also by Sir Clifford Allbutt's case in the same journal March 11th, 1916.

Dr. Ede and others suggested that the fragility of the red cells is increased, but Dr. Humphry and Dr Dorothy Ward found in Sir Allbutt's case that the fragility was not increased but was, in fact normal.

From the appended tables of the Results of Blood Examinations of the cases under observation it will be observed that the red cell counts, before operation was performed, average about 3,675,037 with extremes of 2,870,000 and 5,620,000; the reduction therefore is not extreme. Poikilocytosis is slight and polychromatophilia is absent, but the red cells vary in size microcytes and occasionally megalocytes are present. Although most authors including Osler & Ledingham say that nucleated reds are not often met with, except in very severe and advanced cases yet in three of the cases under observation, the nucleated red cells are persistently present in varying numbers, with extremes of 3 and 39, almost all of which are normoblasts. Of the three cases two of them were splenic anaemia without ascites or jaundice while the other was Banti's disease with jaundice and ascites.

The average haemoglobin is about 60 per cent with extremes of 40 and 93 per cent., so that the reduction is proportionately greater than the red cells making the colour index below normal. In case No. 5. on one occasion the colour



index is above normal. This according to Ledingham sometimes occur in very advanced cases. Case No. 5. was an example of an advanced case.

The average white cell counts is 6,868 with extremes of 1,250 and 15,620. The counts show great variations in number and these variations are seen in the cases where no obvious complications could be detected but seemed to depend on the administration either of arsenic alone or in conjunction with iron. The variation does not only affect the white cells but also the red cells and the haemoglobin proportionately.

In reviewing the differential counts, whether the white cell count is normal, increased or diminished, the finely granular neutrophile polymorphonuclears, in all except one case are reduced in number. They also vary in size. Regarding the percentage of the lymphocytes, small and large inclusive, in cases 1, 2, and 3 they are generally reduced also, but in the advanced cases - Case 4 & 5 they are greatly increased. The transitionals, basophiles and the eosinophiles are all increased. In all except case No. 4. myelocytes are present. The percentage varying from one to fourteen.

Three of the cases were operated on and the spleens removed, unfortunately every one of them developed pneumonia and in one case this went on to empyema. The appended tables show that owing to the above complications neither the red cells nor the haemoglobin improve and there is leucocytosis with the percentage of the finely granular neutrophile poly-

morphonuclears increased. Later the red cells and haemoglobin show improvement and the white cells also showing a tendency to returning to the normal. About nine to twelve months after the operation the lymphocyte percentage seems to be increased. This point was shown by Dr. Ede in his thesis.

The variations of the blood counts, the haemoglobin, & the presence of abnormal red & white cells; the fibrotic condition of the spleen in which there is absence of signs of excessive destruction of blood cells; the effect of arsenic & iron on the blood counts and lastly Professor Mitchell Clarke's suggestion that there is failure in the eliminative function of the spleen, suggest that splenic anaemia or Banti's disease may be due to a disturbance in the erythroblastic and leucoblastic functions of the marrow.

This disturbance of function may be accompanied by the production of a bye product in the nature of a toxin. The spleen being the chief filter of the blood would naturally suffer and undergoes fibrotic changes and thus loses its eliminative function whereby allowing the abnormal cells to circulate in the blood. The enlarged spleen would act mechanically in provoking the condition by causing a general disturbance in the various systems especially the digestive tract & thus impair the general health and also aggravate the disturbance in the marrow until the latter does not respond to the action of iron or arsenic.

Haemorrhages from Mucous Membrane.

Haematemesis is the commonest type of haemorrhage met with in this disease. It is characteristically recurrent & may extend over many years, thus a woman was admitted thirteen times in 15 years for severe haematemesis into the London Hospital (Hutchison). The haemorrhage may be profuse, thus in one of Osler's cases, it was thought that the patient had lost three quarts of blood in thirty six hours. It is the most serious symptom and usually raises the question of splenectomy. According to Sutherland & Burghard, (Royal Society of Medicine, Vol. IV. 1911) haematemesis does not appear to be so common in children as in adults, but Osler has recorded one case.

The bleeding may come from congestion and diapedesis of gastric mucosa, or from erosions of the mucosa due to action of bacteria or according to Osler & Rolleston due to mechanical causes related to the enlarged spleen & not necessarily to cirrhosis of the liver, causing varicosity of the oesophageal veins at cardiac end of stomach and the rupturing of these varices may be the source of the blood.

Other forms of haemorrhage such as Epistaxis, oozing from the gums, haemoptysis, haematuria and less common retinal haemorrhage and ecchymosis in the skin. According to Osler these forms are usually met with in advanced cases with anaemia. Ledingham on the other hand regards epistaxis as the commonest and earliest form of haemorrhage.

Of the five cases under observation two had no history of any haemorrhage. One had frequent slight haemoptysis, another had history of recurrent epistaxis and the last had haemorrhages under the skin.

Pigmentation of Skin. The general nutrition of the patient is usually good and emaciation is usually seen in advanced cases. As a rule pallor is present. A diffuse bronzing or a peculiar steel gray discoloration of the skin, rarely so extreme as to suggest Addison's disease has been described in some cases. In a few cases the pigmentation has been patchy and intensified by an accompanying leucoderma. West suggests that Arsenic may be responsible for the pigmentation in many cases.

Two of the cases under observation had bronzing; deeper on the exposed parts of the body. One was jaundiced and the last had an earthen pallor.

Jaundice. According to Osler moderate jaundice occurs occasionally in association with cirrhosis of the liver or independent of such association, related to circulatory disturbance arising from the enlargement of the spleen or from pressure by the spleen. According to Sutherland & Burghard this jaundice is due to active haemolysis in the spleen. The disappearance after splenectomy certainly is suggestive of this view. One of the cases which had jaundice & had splenectomy performed, the



jaundice did not show any signs of clearing up . The jaundice in this case was undoubtedly due to cirrhosis of liver as this organ was enlarged.

In most cases the liver is of normal size but frequently it is slightly enlarged, one or two inches below the ribs. In advanced cases with the terminal cirrhosis the organ may be contracted and diminished in size.

In three of the five cases under observation the liver was enlarged.

Ascites. In the later stages of the disease with cirrhosis of the liver, ascites may be a prominent feature.

Ascites may occur even in the absence of hepatic cirrhosis, possibly due to irritation of the peritoneum by the enlarged spleen or as a result of the anaemia. Ascites was only present in the two cases which showed evidence of jaundice. Oedema of the feet, of cardiac origin, may occur in the later stages of the disease.

Heart may show signs common to all forms of pronounced anaemia such as fine blowing systolic murmurs, palpitation on exertion, and dilatation. This was present in all the cases under observation.

Lungs. In all the cases under observation either before or after operation there are signs of slight oedema or bronchitis.

Digestive system.

Loss of appetite and constipation are common. Diarrhoea, vomiting and colic may occur but not very prominent. Banti regards that diarrhoea marks the transition from splenic anaemia to Banti's disease.

Urine may show traces of albumin and in rare cases nephritis have been described.

Temperature is usually normal. In advanced cases there may be a tendency to an afternoon rise of temperature 100° F or higher. In cases where there is a known complication the temperature is irregular or hectic. Two of the cases showed a subnormal temperature, one a normal temperature and the other two, one with lung complication and the other was an advanced case showed irregular charts.

Emaciation and Muscular wasting are marked features in the later stages of Banti's disease proper. In one of Hollin's cases the muscular wasting was very prominent. This wasting has been attributed to vaso-motor paresis but according to Hollins it is most probably due to the action of toxin on the central nervous system.

It is agreed upon by most authors that the terms splenic anaemia and Banti's disease are terms used for the different stages of the same disease. The term Splenic anaemia is used for the earlier and non-ascitic stage of the disease and when anaemia is not marked, but leucopenia may be the change.

Whilst Banti's disease is used for the later stage which is characterised by atrophic cirrhosis of liver, ascites and when this stage is reached emaciation and muscular wasting and anaemia are prominent. These being absent in splenic anaemia. Due to the presence of the ascites plus the splenomegaly and the cirrhotic condition of the liver, the digestive and vascular symptoms, which may be present in a slight degree in splenic anaemia, become aggravated. Banti as mentioned above, regards diarrhoea as marking the transition between the non-ascitic and ascitic stage.

#### Diagnosis.

(1) Kala - Azar is a tropical disease and is very rare in this country. It is caused by a parasite of the Leishmania group. The bed bug, the plant feeding bug - conorrhinus - and the dog flea have been looked upon as the transmitting agents of the disease. This disease resembles splenic anaemia in having a large spleen, anaemia, leucopenia and sometimes haemorrhages, but is distinguished by the history of residence in the tropics, the irregular fever especially if there is a double rise in the twenty four hours and to clinch the diagnosis, by the finding of Leishman's Donovan bodies in the splenic cells in a film smear from a splenic puncture.

(2) Malaria is another tropical disease which causes splenomegaly, anaemia, and leucopenia, but is usually

distinguished by history of residence in the tropics, of recurring fevers with the characteristic symptoms of the cold, hot and sweating stages, by the effect of quinine on the fever and lastly by the finding of the parasite in the blood.

(3) Spleno-Medullary Leukaemia. Is a disease characterised by a persistent increase of white cells in the blood, and enlarged spleen and haemorrhages. It may occur at almost any age but is most common in adult life, between the ages of twenty and fifty and affects men more than women; a small number of cases have occurred in infancy.

Symptoms. The onset is insidious, as a rule, attention is often drawn to the condition by progressive enlargement of the abdomen, or by some comparatively insignificant symptom, such as shortness of breath, palpitation, pallor, epistaxis or a pain in the side. Later weakness and loss of flesh may be noticed. Anaemia is not a necessary accompaniment of all stages of the disease; the patient may look very healthy and well.

The spleen is always enlarged. The enlargement is gradual and usually very great, and in many cases the organ occupies fully one half of the abdomen, reaching downwards to the pubes and extending beyond the middle line. The spleen is usually hard in consistency and the anterior border and in some the notch or notches can be distinctly



felt. The size varies, it may be larger after meals and smaller after an attack of diarrhoea or haemorrhage. Pain and tenderness over the splenic tumour are common, but may be absent. On auscultation a murmur is sometimes heard.

The liver is enlarged in some cases. The lymphatic glands may be somewhat enlarged from infiltration of leucocytes. The inguinal and axillary are perhaps the most commonly involved, as anaemia sets in symptoms of cardiac dilatation appear such as cardiac murmurs and oedema. Ascites occur in the terminal stages.

The skin is dry and pale and petechiae are not uncommon. In man priapism is sometimes present and in women the menses may be suppressed. There is usually some fever the temperature may range from  $102^{\circ}$ - $103^{\circ}$  F. Haemorrhages are common and epistaxis is the commonest form. In some cases the haemorrhage may be fatal.

Leukaemic retinitis is a part of the haemorrhagic manifestation. From the above description, the similarity between spleno-medullary leukaemia and splenic anaemia is striking and it is impossible to differentiate the two without a blood count.

The striking change in the blood of spleno-medullary leukaemia is an increase in the colourless corpuscles. The usual limits are from 200,000 to 500,000 per c. mm. but may reach 1,000,000 or more. In Osler's

series of cases the average was 298,700 per c. mm. and the average ratio to the red cells was 1 to 10.

During remissions and during intercurrent affections the leucocyte count may fall to a low figure, but even in these cases abnormal cells persist. The increase is in all the forms, but the chief feature is the presence of a large number of myelocytes, which range from 30 to 50 per cent.

The red cell count may be normal, the cells not altering in shape, size or staining property but when anaemia sets in the count may fall to 2,000,000 per c. mm. and the cells show changes in the size, shape and staining property. In the majority of cases nucleated reds, normoblasts and megaloblasts are present even from the outset of the disease. Before anaemia sets in the haemoglobin remains proportional to the number of red cells, but on the onset of anaemia the haemoglobin is proportionately greater diminished and thus a chlorotic type of anaemia resulted.

(4) Gaucher's Disease - Primary Endothelioma of the Spleen.

According to Stengel, whose opinion is generally accepted, this affection is a type of new growth, an endothelioma and it differs entirely from splenic anaemia. It is often a familial disease, and affects

children more than adults; females more than males. As regards symptoms and course the disease closely resembles splenic anaemia and Banti's disease.

Pathology. The spleen is uniformly enlarged and retains its usual contour. On section the splenic substance presents a grayish red appearance with scattered white patches and streaks. Malphigian bodies are small. Histologically the section shows large hyaline cells from 20 to 40u in size, with small nuclei filling the alveolar spaces. These characteristic 'Gaucher cells' are also found in the enlarged liver, lymphatic glands and bone marrow. Yellow pigment may also be found in the above organs. The symptoms are those of splenic anaemia or Banti's disease with the exception that jaundice and ascites are rare. A brownish yellow wedge shaped thickening of the conjunctiva, beginning at the nasal side of the cornea, is said to be present in Gaucher's disease. Regarding the blood changes, the red cells may be diminished to three millions, haemoglobin more diminished and normoblasts may be found. The white cells are either unaffected or there may be a slight leucopenia. This affection may be distinguished from splenic anaemia by its incidence in children, its familial occurrence, the eye symptom and the absence of ascites and pathologically by the finding of the characteristic Gaucher's cells in the spleen, liver, marrow and lymphatic glands.

(5) Splenomegaly with Acholuric Jaundice. First described by Minkowski and sometimes called after his name. It is a familial often hereditary disease, characterised by anaemia, icterus and splenomegaly.

Pathology. The spleen was enlarged in all recorded cases. Fibrosis and perisplenitis may occur but the chief change is simple engorgement and pigmentation mainly in the endothelial cells. Liver and kidney show no special changes, except pigmentation may be present.

Symptoms. The characteristic features of the disease are (1) Its comparatively slight effect on the general health. (2) Enlargement of the spleen is an early symptom. The enlargement is chronic and in time becomes very hard. (3) Chronic slight jaundice, its intensity varies from time to time. It seldom becomes so deep as in obstructive jaundice but the conjunctivae are usually definitely coloured and bile pigment can be demonstrated in the blood serum. The stools are well coloured and there is usually absence of bile pigments in the urine, but urobilin is usually present. In a few cases gallstone colic has been present, due to the presence of small calculi.

Epistaxis is a symptom of fair frequency. Retinal haemorrhages and purpuric spots have been recorded in one instance.



Blood changes. The anaemia is of the chlorotic type. The red cells are usually reduced in number to two or even one million. Anisocytosis is a marked feature. The microcytes may be looked upon as one of the essential features of the disease. Poikilocytosis, polychromasia, punctate basophilia and nucleated reds may be present.

The white cell count shows no special abnormality. Myelocytes in small number are generally found.

Chauffard showed that in this affection the red blood corpuscles show increased fragility. Blood serum contains bile. Cures have been reported after splenectomy.

This affection is distinguished from splenic anaemia or Banti's disease by the following points:-

1st Its familial or hereditary tendency.

2nd The good health of the patients who are usually children.

3rd Presence of prolonged intractable jaundice.

4th The condition of the urine.

5th The increased fragility of the red blood cells.

(6) Syphilitic Splenomegaly.

As a result of syphilis enlargement of the spleen may be due to gumma or lardaceous disease, or it may follow on gummosis hepatitis either congenital or acquired. In the later stages this condition is very similar to Banti's

disease as anaemia, slight jaundice, ascites, splenomegaly and recurring haemorrhages occur. For the diagnosis of this condition we have to rely on the occurrence of other syphilitic manifestations, nodular character of the spleen and liver, history, Wasserman test and on the effect of antisyphilitic treatment.

(7) Pernicious Anaemia or Addison's Anaemia has been described as a progressive, recurring, profound and usually fatal anaemia of unknown origin, characterised by haemolysis and imperfect action of the blood forming organs.

No age is exempt but the disease is most common between thirty and fifty, Osler reported a case of a boy of ten years old. Hereditary and familial tendency sometimes occur. The disease is more common in males than females. William Hunter described the condition as a chronic febrile infection due to auto-intoxication from the gastro-intestinal tract and produced by an organism the toxins of which give rise to haemolysis. There are a host of other conditions and diseases which have been suggested as casual agents in pernicious anaemia, but the majority of them are not proven.

#### Pathology.

Tongue. Hunter paid much attention to the smooth atrophic looking areas on the tongue during life, but no microscopic change to account for these have been found.

Stomach and intestines may show atrophy and thinness of the coats, smoothness of the mucous membrane and atrophy of the glands. Liver is always fatty and may be enlarged and is rich in iron. The haemosiderin pigment occupies the outer & middle zones of the lobules.

Spleen is enlarged in some cases. No constant change is found in the malphigian bodies. The pulp is congested & cells present, vary in character, polymorphs, lymphocytes, large phagocytic cells containing red blood cells, giant cells, basophil cells and nucleated reds may be present. Pigment is present in varying amount in the pulp and some in the endothelial cells and leucocytes. Some of this pigment gives the iron reaction.

Kidneys usually show catarrhal or interstitial nephritis & the cells in the convoluted tubules may contain pigment giving the iron reaction.

Heart is the seat of advanced fatty degeneration, so that the "thrush's breast" appearance may be seen on the endocardium especially of the ventricles.

Haemolymph glands are usually large, and dark red in colour and contain pigment.

The bone-marrow is red and contains large numbers of megaloblasts and giantoblasts, suggesting a compensatory hypertrophy. In few cases it is atrophied, (Aplastic anaemia).

Nervous system, small haemorrhages may be present in the membranes brain & cord. In the white matter of the cord small foci of sclerosis have been seen. Combined postero-lateral sclerosis is the common lesion. The peripheral nerves may show indications of neuritis.

The presence of large quantities of haemosiderin in the liver, and of pathological urobilin in the urine, are strongly in favour of the haemolytic origin of the disease.

#### Symptoms.

The onset is always insidious. Although muscular weakness is the usual complaint which causes the patient to seek medical advice yet it is remarkable how much work a patient with very low blood counts can do. The patient usually presents a plump appearance and in a typical case the skin has a peculiar waxy lemon-yellow tint, has a velvety feeling. In a small proportion of cases a definite icterus in the skin and conjunctivae is seen. Pigmentation of the skin and small petechae may be present.

Haemorrhages are frequent, especially retinal haemorrhages, haemoglobinuria and haematuria. Epistaxis & melaena may also occur.

Alimentary system. Loss of appetite, sensation of pain or discomfort in the epigastrium, sickness, vomiting and diarrhoea may be present. Enlargement of the liver is only seen in chronic cases & the enlargement is not great



and it comes and goes with successive attacks and remissions.

The Spleen is firm but is not usually enlarged. In some cases the spleen is enlarged, but the enlargement very seldom extends beyond one inch below the costal margin. The enlargement comes and goes as the liver does.

circulatory symptoms such as palpitation, shortness of breath, faintness, cardiac murmurs and Bruit de diable and oedema may occur.

Urine in all respects show little abnormality except for the presence of urobilin in excess as pointed by Hunter & Mott.

Nervous system. Numbness and tingling are common, sometimes marked neuritic pains may be present. Symptoms of postero-lateral or lateral sclerosis may occur. Neuresthenia, depression & delusions may be present but not marked.

Blood changes. The total quantity in the body is much diminished. The red blood cells are greatly diminished. An average estimation when patients come under observation would be about 1,575,000.

Gabot says that in no other disease is the red cell so often reduced to below two millions. In 12 per cent. of Osler's cases the count was under one million. The lowest count on record is in a patient of Quinke's 143,000 per c. mm. The haemoglobin although quantitatively reduced, is relatively high so that the colour index is almost always above 1. Poikilocytosis is very marked, some of the cells

are elongated, rod like, others pyriform.

Anisocytosis is also remarkable; megalocytes measuring 8, 11, or even 15u and microcytes from 2 to 6u in diameter are numerous.

Laache showed, that the presence of megalocytes is one of the most pathognomonic features of the disease.

Polychromatophilia is also common. Normoblasts and megaloblasts are constantly present, varying very much in number from day to day. A large number of them appear during the crisis. The white cells are generally normal or reduced in number. Polynuclear cells are rarely reduced. Myelocytes are frequently present even up to 8 to 10 per cent.

To distinguish this from spleen anaemia or Banti's disease, the following points must be relied upon.

1st. Spleen is usually much smaller than in  
Splenic anaemia.

2nd. The character of the blood film, especially  
the presence of megaloblasts & microblasts  
and more important still of megalocytes &  
microcytes.

3rd. The high colour index.

8. Haemochromatosis is a disorder of metabolism characterised by the deposition of haemosiderin pigment in the glandular organs, and by a progressive sclerosis of various

organs and in a large proportion of cases it is associated with diabetes. Out of 60 cases only one occurred in a woman. Middle aged men are mostly affected.

Pathology.

The marked feature is the bronzing of the various organs. Liver is enlarged and sclerosed. Spleen is also enlarged & sclerosed. Pancreas is atrophied and sclerosed. Lymphatic glands are also pigmented. The pigment is haemosiderin, some of it is haemofuscin - non-iron reacting.

Clinically most of the cases are associated with diabetes, and present bronzing of the skin, this varies from dark brown to bluish black, weakness, enlarged liver and spleen, but there is no anaemia or any special changes in the blood.

We depend on the absence of anaemia and the association of sugar in the urine in differentiating this malady from splenic anaemia or Banti's disease.

9. Gastric ulcer usually occurs in young & chlorotic women who present the definite digestive symptoms. The absence of these symptoms & the presence of enlarged spleen, which is absent in gastric ulcer, differentiates this from splenic anaemia.

10. Thrombosis or occlusion of the Splenic vein may set up a sequence of symptoms which form an exact imitation of splenic anaemia or Banti's disease viz., splenomegaly,

Anaemia, leucopenia and haematemesis . It is impossible except in a case presenting a history of injury followed by the onset of the above symptoms, to distinguish it from splenic anaemia or Banti's disease.

11. Portal cirrhosis of Liver is most common in the middle period of life, in the male sex, and in those addicted to alcohol especially spirits. In the late stage of the disease when the liver is atrophied, anaemia, wasting, haemorrhages, enlarged spleen and ascites occur and thus simulates Banti's disease very closely.

#### Pathology.

The liver may be enlarged but more commonly it is reduced in size. The enlargement may be due to active congestion and therefore temporary, passing into atrophy at a later stage, or it may be due to extensive fatty change, when it is permanent. The smaller the liver the rougher & more nodular will the surface be and conversely the larger the liver the greater is the probability of its being smooth or but slightly roughened.

Microscopically the dense sclerosis is seen to be going on chiefly in the periphery & encircling several lobules, whose cells are degenerated or undergoing degeneration. This fibrosis obstructs the portal circulation and later, in some cases, the biliary ducts. The result of the former is the establishment of collateral circulation. The most common



anastomosis are between:-

- (1). Gastric and oesophageal veins producing the oesophageal varix which when ruptured produce haemetemesis.
- (2). The superior haemorrhoidal of the inferior mesenteric and the inferior haemorrhoidal of the internal iliac, producing piles and the rupturing of these is a source of bleeding from the bowels.
- (3) The veins of the intestines and the retroperitoneal veins.
- (4) The accessory vein of Sappey & branches of the epigastric veins near the navel producing the characteristic "Caput medusa".

#### Symptoms.

So long as collateral circulation is maintained cirrhosis may exist without any symptoms.

At the beginning there may be symptoms pointing to catarrhal condition of the stomach such as loss of appetite, acid eructations, and vomiting. Epistaxis is common in the early stages, sooner or later haemetemesis or oesophageal haemorrhage may occur. Haemorrhages from the bowels may occur for several years without haemetemesis. Sense of weight or pain in the hepatic region may be complained of. The liver in this early stage is usually enlarged & tender. Later in the disease, emaciation, pallor and enlargement of the spleen occur. The splenomegaly is not great. The

liver is usually diminished & the surface rough. As time wears on Ascites and emaciation come to be the prominent feature. Jaundice is late and often slight, sometimes it is absent throughout. In the terminal stages drowsiness, delirium and coma may supervene and lead to death. The anaemia is of the chlorotic type. The differentiation of this from Banti's disease is very difficult. The history of the case carefully taken may be the only means by which a correct diagnosis may be arrived at. Some cases which have been diagnosed as Banti's disease have ultimately turned out to be examples of cirrhosis of liver.

#### Treatment.

Splenectomy seems to be the only treatment which holds out any hope of permanent cure, but before the operation be proceeded with, a definite diagnosis must be made and some time may be needed for this. During this period hygienic, medicinal and X-ray treatments may be resorted to, although no permanent benefit or cure can be obtained from the above measures.

#### Hygienic.

Rest in bed is strongly indicated in cases where the weights of the spleen causes discomfort and dyspnoea on exertion, when the anaemia is marked and when there is fever, and when the Banti disease proper stage is reached. Free exposure to the vivifying influence of sunlight

and good air is one of the best blood restoratives we have. Mental worry and exposure to inclement weather especially chills must be avoided.

Unhealthy conditions of the gums and teeth, which undoubtedly may exert material influence on the production and maintenance of anaemia, must be treated at once and thoroughly by appropriate local measures. Brushing of the teeth night and morning with some antiseptic lotion or powder should be insisted upon.

Diet.

Defective or unsuitable food would certainly aggravate the already existing anaemia and if this is not present, it would certainly produce it. An exclusive non-introgenous diet has been proved to diminish the haemoglobin, while this is augmented by a diet rich in albuminates.

At first the diet should be small or moderate in quantity, so as not to overtax the feeble digestive powers, and it should be in a readily digestible form. Milk, when easily digested, and cream, and all forms of animal food, presented in a form easy of digestion, are useful. Raw or slightly cooked meat, reduced to a pulp and flavoured, is of value as a blood restorer, because of its high percentage of iron. If it is necessary a few grains of pepsin or dilute hydrochloric acid may be added

to aid digestion.

Besides the above, some form of digestible fat in the form of butter or cod liver oil should be added especially when wasting is present.

In severe cases where the digestive powers will not allow the above diet, or where cardiac failure is present, the yolks of two or three eggs, beaten up with a little boiling water and flavoured with a little sugar and with a teaspoonful or two of brandy added, produce an excellent concentrated form of nourishment. The value of the yolk of an egg is that it is easily digested and is rich in iron.

A little spirit or wine with or without water is sometimes useful, both as a stimulant and a sedative.

#### Medicinal.

Drugs, as mentioned above, are of little avail in bringing about a permanent cure. It is doubtful if there is on record where Splenic anaemia or Banti's disease has been cured by drugs only. The condition of the blood may be temporarily improved by drugs, but no drugs that are known can diminish the splenic enlargement. This being so, drugs are invaluable during the period spent for the diagnosis to be made, as the temporary improvement of the blood is usually followed by an improvement in the general condition of the patient, which is essential if the splenectomy is to be withstood successfully.

Arsenic and Iron are the two drugs, which are



usually advocated for in splenic anaemia or Banti's disease. Both drugs are well known haematinics and tonics. The improvement in the blood condition is well illustrated in the appended charts, showing the results of Blood examinations. The improvement was in all respects, thus the red cells, nucleated reds, white cells and haemoglobin were all increased. This improvement was also noticed in Case No.1 and No.3 who had arsenic only while the other cases had iron combined with arsenic. In the former cases the arsenic was given in the form of Liquor Arsenicalis  $m \frac{iii}{iii}$  to  $m\bar{x}$ , while in the latter cases the prescription was as follows:-

Ferri et Ammonii Citratis gr.  $\bar{v}$

Liquoris Arsenicalis  $m \frac{iii}{iii}$  increased gradually to  $m\bar{x}$ .

Aquae

Ter in die post cibos.

Some cases show no improvement at all with iron or arsenic given separately or in combination. As in other anaemias gastro-intestinal disturbance is usually present and the administration of aperients should be carried out before proceeding with iron or arsenic.

Barr - Lancet, August 23, 1902 recommends Iron perchloride and Calcium Chloride as valuable haematinics.

Salol or Beta Napthol which are intestinal anti-septics are given by those who regard the source of the toxin to be in the gastro-intestinal tract.

X-ray treatment may also produce temporary relief in some but not in all cases. The exposure is usually over the spleen, either once or twice weekly. By this treatment the spleen has been reduced in size, and Gulland has reported a case where the blood became normal and the spleen returned to its normal size with X-ray treatment.

In the cases under observation no appreciable improvement either in the blood or size of spleen was noticed.

Mr. H. S. Scuttar suggested that the risks of laparotomy and splenectomy might be avoided and good results obtained by inserting a tube of radium by means of a specially constructed trocar directly through the abdominal wall into the substance of the spleen.

#### Operative.

Splenectomy is the selective operation, and when Splenic Anaemia or Banti's disease has been definitely diagnosed and if the patient's condition is satisfactory the operation should be advised, for the following reasons, 1st. It is generally agreed that the disease is essentially and without exception progressive and tends ultimately to destroy life. Secondly, that no treatment other than splenectomy will produce a permanent relief and also obviate the inevitable tendency to cirrhosis and lastly, that splenectomy is usually followed by an immediate improvement even in advanced cases and it is in the general opinion that the improvement is permanent.

The earlier in the disease the operation is performed the greater the prospect of a satisfactory and permanent recovery is. The result of the operation is very encouraging as the blood may return to normal and the general health of the patient improves almost immediately in early and uncomplicated cases.

According to Mayo - Lancet, November 25th 1916, cases with splenomegaly, advanced cirrhosis of liver and ascites were restored to health; one of his cases is alive now for more than seven years, (1916).

The possible causes of death from the operation are haemorrhage at the time, this can usually be obviated if among other essentials in the operative technique, perfect access for its control is afforded; gastro-intestinal haemorrhage after a few days, due perhaps to thrombosis of the splenic and other larger veins, but in cases operated upon betimes and under favourable conditions this event also is far less frequent; shock pneumonia and vague gastro-intestinal symptoms some months after the operation.

According to Dr. E. Graham, splenectomy is both useless and dangerous in cases where the haemoglobin is below 30% and the red below 2,000,000.

With regard to the result of the operation I shall now append the various published statistics.

In the Annals of Surgery XXIV 1901,

19 cases were reported with 14 recoveries, 4 deaths, &  
1 not reported, making a mortality of 21%.

In the British Med: Journ: 1906 Armstrong collected together

32 cases operated on with 22 recoveries, 9 deaths,  
1 unrelieved making a mortality of 31%.

In the Annals of Surgery XLVIII 50, 1908 Johnston reported

61 cases operated on with 49 recoveries, 12 deaths,  
making a mortality of 19.6%.

Sir Clifford Allbutt and his colleagues in the British Medical Journal March 11 1916, say "That the issue of the present series of operations is to show that the advantage of operating in an early and uncomplicated stage of the disease is enormous; the mortality is thus reduced in cases of this class from about 50 to about 10 per cent. Banti's figures are to the same effect." With regard to the mortality after operation of Banti's disease proper, the mortality is naturally much higher. Banti gave as his figure 50 per cent while Rodman and Willard - Annals of Surgery 1913 - gave theirs as 56.25 per cent.

Jaffé in the Zentralblatt für Chirurgie, No.28, 1906, & Mr. Hey Groves in The Bristol Medico-Chirurgical Journal, p. 331, December 1913 suggest that in the presence of well marked atrophic cirrhosis of the liver and ascites, the



splenectomy may be followed by Talma's operation.

Dr. Edwin E. Graham of Philadelphia, in The American Journal of Obstetrics, September 1916, says "That until the year 1908, the mortality following splenectomy for splenic anaemia was 17 per cent. from 1908 to 1912, forty-seven splenectomies were performed with five deaths, this mortality being a little above 10 per cent. But these figures were based on cases in which the symptom-complex of Banti's disease was not present".

Mayo has reported eighteen cases of splenectomy for splenic anaemia without a death.

I shall now endeavour to tabulate the results of splenectomy for splenic anaemia & Banti's disease since 1912.

- |       |  |  |
|-------|--|--|
| 1913. | Mr. Hey Groves,<br>Bristol Medico-<br>chirurgical Journal,<br>Dec. 1913. Vol. XXXI.) | ) Woman aet 40. Resulted<br>with in<br>Banti's Recovery.<br>disease.                 |
| 1914. | Dr. Essex Wynter &<br>Sir Bland Sutton.<br>Lancet, Nov. 28. 1914.)                   | ) Boy aet. 13.<br>with Resulted<br>splenic in<br>Anaemia. recovery.                  |
| 1915. | Mr. Gilbert Barling -<br>Lancet, Jan. 30. 1915.)                                     | Man aet 19.  |
|       | (1)  | with Resulted<br>Splenic in<br>Anaemia recovery.                                     |
|       | (2)  | Man aet. 20. Resulted<br>with<br>Splenic not<br>Anaemia & Satisfactory.<br>Jaundice. |

(41)

- (3) Girl aet 6 ( Resulted  
with ( in  
Splenic ( Death  
Anaemia. ( from  
( Shock.
1916. Dr. Edwin Graham - American ) Girl aet 7 Resulted  
Journal of Obstetrics, ) with in  
Sept. 1916. ) Splenic Recovery.  
Anaemia.
- Mr. Hey Groves - Case No. 1. ) Man. aet 29.  
under observation at ) with Resulted  
General Hospital, ) Splenic in  
Bristol. ) Anaemia. recovery.
- Mr. R. P. Lansdowne, Case ) Woman aet. 36.  
No. 2. under observat- ) with Resulted  
ion, General Hospital, ) splenic in  
Bristol. ) Anaemia. recovery.
- Sir Clifford Allbutt, British ) Woman aet 45 Resulted  
Medical Journal, ) with in  
March 11th, 1916. ) Splenic Recovery.  
Anaemia.
- Mr. Cholmeley, British Med. ) (1) Boy aet 6.  
Journal, June 10. 1916. ) with Resulted  
Splenic in  
Anaemia Recovery.
- (2) Boy aet. 12.  
with Resulted  
Splenic in  
Anaemia, recovery.
1917. Mr. R. P. Lansdowne, Case ) Woman aet 30.  
No. 3 under observat- ) with Resulted  
ion at General Hospital, ) Banti's in  
Bristol. ) disease. recovery.
- Mr. G. Grey Turner, ) Report 3  
Practitioner, June 1917 ) cases of Splenic ) All re-  
p. 511. ) anaemia & 1 case ) covered.  
of Banti's.)

1918.	Mr. Footner &	)	Black Girl aet 19.	
	Mr. R. Archibald. Lancet,	)	with Banti's	Resulted
	Jan. 19. 1918.	)	disease.	in
				recovery.

Out of the seventeen cases recorded from 1913 to 1918, there are five Banti's diseases & 12 Splenic anaemias. The result of the operations is most gratifying for there is only one death out of the seventeen cases making the percentage mortality to about 5.8 per cent.

#### Epiplopexy.

According to Edwin E. Graham of Philadelphia in The American Journal of Obstetrics, Sept. 1916, the operation of choice in Banti's disease is Talma's operation. This procedure would seem to be merely palliative in this affection for if the spleen is regarded as the cause of the disease, as long as it is left the disease must be progressive in spite of the Talma's operation, the object of which is to relieve the ascites by increasing and improving the collateral circulation between the portal and systemic circulation.

Karl Eberly in the Journal of the American Medical Association, Vol. LXVII, No. 1, July 1, 1916, reports a case of a woman aet 63 with Banti's disease, who died from gastrointestinal haemorrhage after rallying well from the operation and progressing nicely for several days.

Dr. Murrell in The Medical Press and Circular, Jan. 8. 1902 reported a girl aet. 20 suffering from Banti's disease on whom Mr. Spencer performed Epiploexy. This patient showed temporary relief but died about one & a half year after with recurrence of ascites.

From the above cases it would seem that this operation is only palliative but not curative as splenectomy is in this affection.

With regard to the treatment of the five cases under observation, during the interval spent in making the diagnosis, for most of the time the patients were kept at rest, their teeth were attended to by the Dental Surgeon. With regard to medicine iron and arsenic were the drugs used either separately or in combination. Salol was also given as an intestinal antiseptic. As most of the patients suffer from coughing and had slight bronchitis limited to the bases of the lungs, alkaline expectorants were administered. Fresh air and sunshine were also allowed to the patients as the weather permitted. All of the cases had X-ray treatment some once and others twice weekly. Three of the cases showed temporary relief and splenectomy had to be resorted to. One of the cases did not show any improvement and later developed ? pulmonary tuberculosis of one lung and died outside hospital. The other case showed no improvement and



refused operation and went home but lived only for three months after she left hospital.

Salvarsan has been used in some cases but its effect is also temporary, also some cases are greatly benefitted by it.

Summary and Conclusion.

The etiology of the disease is still unknown.

The following points:-

1st. Two of the cases under observation had history of vague pains in their limbs which led to the diagnosis of rheumatism in one case and in the other neurasthenia.

2nd. The variability of the blood counts, brought about by the administration of arsenic or iron, before and after operation.

3rd. The persistent presence in the blood of abnormal red & white cells.

4th. The fibrotic change in the spleen in which there is absence of signs of excessive destruction of blood cells.

5th. Professor Michell Clarke's suggestion that there is failure of the eliminative function of the spleen.

6th. The hyperplastic changes of the haemolymph glands lying along the thoracic aorta and in the retroperitoneal region, as described by Dock, Warthin and Stengel & thought by them to be compensatory for the loss of the haemolytic function of the spleen.

Suggest that the marrow may be the primary seat of lesion in splenic anaemia or Banti's disease.

As to the cause of the disturbance in the marrow it is difficult to say. Two of the cases under observation

had previous history of Enteric fever, one of Malaria and appendicitis, one of severe abdominal injury and the last had no severe illness or injury.

With regard to the symptoms all the cases under observation present the characteristic symptom of splenic anaemia and two of Banti's disease proper. All of the cases had evidence of bronchitis in the bases of the lungs especially of the left, this probably is due to the irritation or compression caused by the enlarged spleen. One of the cases showed telangiectasis varying in size from that of a sixpenny piece to a shilling. The blood pictures show a persistent presence of nucleated red cells and abnormal whites especially myelocytes.

The blood counts for red and white cells and the haemoglobin show variation, under the administration of arsenic and iron, in splenic anaemia but not in the later stage or Banti's disease.

Splenectomy gives relief with a probable permanent cure.

Considering the lung condition of these cases, however slight they may be and the complication which unfortunately occurred in the cases operated on ether anaesthesia should be avoided.

Conclusion.

From the cases under observation, - Cases 1 & 2 diagnosed as splenic anaemia; cases 3 & 5 diagnosed as Banti's disease; we note the close similarity of the differential counts. The presence of nucleated reds mostly normoblasts, and of myelocytes; and the increase in the transitionals, mast cells and eosinophile cells, in all these four cases would suggest that these affections have after all quite a distinctive blood picture which no writer has described. This view is substantiated by the absence of this characteristic picture in case No. 4, which from the history and symptoms was improbable to be splenic anaemia or Banti's disease.

With regard to treatment there is but one and this is Splenectomy without which the disease is progressive and fatal. In case No. 3, this procedure was sufficient to prevent the recurrence of ascites.

Mr. Hey Groves recently told me that the case which he described in The Bristol Medico-surgical Journal, Dec. 1913, p. 331, as having recurring ascites after splenectomy and which required frequent tapping has now quite recovered, no recurrence of ascites occurred, although the Talma-Morrison operation which he suggested might be useful for the relief of the ascites was not performed.



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Case No. 1.

Alexander Rawlins, aged 28, blacksmith by occupation was admitted to the General Hospital, Bristol, under the care of Dr. Michell Clarke, on March 27th, 1916, complaining of an enlarged spleen.

Family History.

Father is alive and well. Mother died of blood-poisoning. Four brothers are alive and well. Two sisters are alive and well. Wife is alive and well, has two children who are very healthy; and never had a miscarriage. No consumption in the family.

Previous History.

When sixteen years of age had a severe blow on the abdomen, which caused him to pass dark blood per rectum for the following two days, after which no more haemorrhage was noticed. He was kept in bed for two weeks although he suffered no pain or saw any blood. For the following two or three years after, colicky pain in the abdomen occurred occasionally. After this he has been in very good health until this present illness occurred, except for shortness of breath and weakness since July 1913. He had no other serious illness, and although he was in Australia he never had malaria.

He denies venereal history.

Habit.

Has been in Australia in a Poultry farm during the last few years.

Has never touched alcohol nor tobacco.

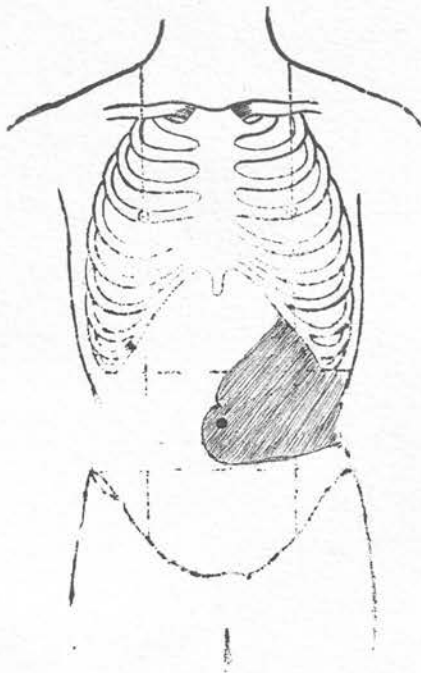


Present Illness.

Present illness began in May 1915, when he suddenly developed diarrhoea which caused him to go to stools about twelve times a day. Had no tenesmus or straining at stools, but a pain across the upper part of the abdomen. The stools contained no mucous or blood but simple loose actions. This continued for two months without treatment and as he was becoming weak, he went to hospital in South Australia in July 1915. There his diarrhoea ceased and the doctor found the enlarged spleen. Has been free from the abdominal pain since July 1915 and thinks his spleen has not further enlarged. Suffers from shortness of breath and palpitation on exertion. Feels weak and easily tired out. Has never had epistaxis or haemorrhages of any kind except for the melaena which occurred sixteen years ago. His appetite is fairly good, bowels are regular, and sleeps fairly well.

Condition on Examination.

Is not emaciated or anaemic, but there is slight bronzing of the face, neck, forearms and hands, probably due to sun burn. There is no evidence of pigmentation elsewhere, no icterus, and the lymphatic glands are not enlarged. No oedema is present. Tongue is pale, flabby and moist. Teeth are in good condition. Temperature is 97.8° F., Respiration is 24. Pulse is 80, regular in force and rhythm.



Case No 1

Abdomen is distended, more on the left than the right side. There is no rigidity, tenderness or ascites. No veins visible. Liver is not enlarged.

Spleen is greatly enlarged, extending forwards to about two inches to the right of the umbilicus and downwards to about seven inches from the costal margin in the left mammary line. It is hard and not tender. Surface is smooth and the border is sharp. A notch can be distinctly made out, just to the right of the median line and above the umbilicus.

There is no resonant note over the tumour.

CHEST. Is well developed and moves freely and regularly on respiration. The lungs are resonant all over. The breath sounds are vesicular with no accompaniments. Vocal resonance and fremitus are normal.

Heart. The apex beat is not visible but palpable in 5th space internal to nipple line.

	Upper border 3rd R.
Right sternal border.	Apex in 5th space. Internal to nipple line and is $3\frac{1}{2}$ " from mid line.

Sounds are clear, regular in force and rhythm. No murmurs can be detected.

Nervous System.

Memory and intelligence are good.

Pupils are equal and react to light and accomodation.

No optic changes can be seen on opthalmoscopic examination.

Abdominal reflexes are present.

Knee Jerks are present and equal.

There is no Babinski or ankle clonus.

No evidence of pareisis or paralysis is present.

Urine. S. G. 1025; acid; shows a deposit of urates and phosphate; no albumin, sugar or bile, or blood.

Faeces - Shows no occult blood.

Blood. Wasserman Test is negative.

No parasites are found in the blood films.

The result of the various blood examinations is given in the appended chart.

The prominent feature of the blood is the presence of a number of abnormal red and white corpuscles, and also the intermittency brought about by the effect of arsenic.

Slight poikilocytosis, and active mitosis in the nuclei of many cells are present.

From the time of admittance onwards the temperature has been mostly subnormal, as shown in the accompanying chart.



Except for the feeling of weight in the left side of the abdomen, slight weakness; conjunctivitis and sore throat which are due probably to the arsenic, patient presented no other symptoms during his nine weeks' stay in hospital. No haemorrhages of any kind, no diarrhoea, no pyrexia occurred. In spite of X-ray treatment the enlarged spleen showed no appreciable diminution in size.

Owing to the uncertainty of the differential counts brought about by the presence of a large number of abnormal white cells especially the transitionals which amounted from 19.6 to 27.2 per cent., and taking into consideration that patient was putting on weight, in the absence of ascites and oedema, and the general improvement, it was decided that it would be wise to wait and keep the patient under observation with the hope that the blood picture would settle down definitely. The patient was discharged on the 26th May 1916, and made an out-patient to follow up the treatment.

#### Treatment.

Rest in bed for first week and after this was allowed up to sit quietly in the fresh air. Ordinary diet was given from the very beginning.

Liquor Arsenicalis m. ~~iii~~<sup>...</sup> three times a day was ordered on the 27th March 1916. This was increased to m V on the 6th April 1916, and to mVI on the 17th April. On the 12th April it had to be discontinued owing to sore throat

and conjunctivitis. It was resumed again on the 4th May 1916 but had to be stopped again on the 22nd May owing to the same symptoms. The effect of the drug on the blood is shown on the appended chart of Blood Examination.

On the 14th April 1916 patient was given X-ray treatment twice weekly.

After carrying out the same treatment as out-patient for three months and the condition of the blood and spleen showed no improvement, and the general condition deteriorating it was decided to re-admit the patient. This was done on August 31st 1916. The condition of the patient was practically the same as before, except that he was weaker, developed a slight cough with no expectoration, easily tired out and has lost weight. The spleen was slightly diminished in size, but the blood was still indefinite. The basis of the lungs were slightly dull and occasional rales could be heard. No ascites or oedema was present. On October 4th, 5th & 6th the temperature which had been subnormal since readmission rose in the evenings, to  $101.4^{\circ}$  F,  $102.2^{\circ}$  F, and  $100.2^{\circ}$  F, respectively but settled down to the subnormal type subsequently. During this rise of temperature pain over the spleen was present and the organ was larger, harder and tender. The latter disappeared when the temperature settled down again. As the general condition of the patient had improved and the lungs had cleared up it was decided that

splenectomy should be resorted to.

On October 16th 1916 Mr. Hey Groves removed the spleen. The organ was removed with ease as there was no adhesions and the pedicle was easily secured. The splenic vessels were tortuous and elongated. The liver was not enlarged or diminished in size. The removed organ measured  $16\frac{3}{4}$ " X  $9\frac{1}{2}$ " X  $4\frac{1}{2}$ " and weighed 4 lbs. 5 ozs. A culture was made from the spleen but no growth resulted. For two days after the operation the condition was very satisfactory but on the third day the temperature rose in the evening to  $99.6^{\circ}$  F, and this rise was maintained for three days and was probably due to constipation. After being normal for two days the temperature suddenly rose again on the 26th Oct., 1916, and assumed a hectic type. The pulse and respiration were also increased. The patient felt unwell and had diarrhoea. On examination it was found that the operation wound was quite clean; the abdomen slightly distended but not tender; the bases of the lungs were diminished in resonance and the entrance of air poor. On Oct. 31st patient had a rigor which lasted ten minutes and this was followed by profuse perspiration, the temperature at the time being  $104^{\circ}$ F, The left base of the lung was dull, with diminution of breath sounds and vocal resonance. A series of Antistreptococci serum was given subcutaneously which produced marked improvement but the temperature was

still swinging although it was coming down gradually. The left base was still dull and on November 26th, 1916 pus was found on exploration. On the same day resection of the rib was performed. A drachm of pus and three quarter pint of sanseous fluid was let out. Pus was examined and was found to contain Bacillus Proteus, no cocci were detected. Since then the temperature has settled down again to the sub-normal type as before and the general health of the patient improved greatly. Before being discharged on the 8th Jan. 1917 patient was very much improved in health and had put on weight.

Seen a year after the operation - October 7th 1917, patient was quite well & feeling stronger but felt weak now and again only. On February 4th, 1918 patient was looking very well and said that he was strong and suffered from no symptoms whatever.

He is now working sixteen hours a day as a gardner.

From the appended blood chart it will be seen that before the rib was resected, the red blood cells were diminished in number, the haemoglobin, low, and leucocytosis present. The increase was chiefly in the finely granular polymorphonuclears, whilst the later blood counts showed a marked improvement in the red cells and haemoglobin, although the abnormal cells were still present in great number.



This case presents the characteristic symptoms of splenic anaemia.

An interesting point is the onset of the case with diarrhoea which Banti regarded as the important symptom indicating the transition of splenic anaemia into Banti's disease proper. The blood is interesting owing to the presence of abnormal red and white cells before and after splenectomy and also in the manner in which it varies on the administration of arsenic.

The beneficial effect of Splenectomy.

Case No. 2.

Mrs A. Long, aged 36, was admitted to the General Hospital, Bristol, under the care of Dr. Michell Clarke, on August 14th 1916, complaining of pain in the left side of stomach and back, and shortness of breath on exertion.

Family History.

Father is alive and suffers from trouble with his water. Mother died of heart disease and paralysis. Four brothers, two of them are alive and well; one died when two months old and the other died of pneumonia. Two sisters are alive and well. Husband is alive and well. Four children are alive and well except that one of them suffers from fits. No tuberculosis is in the family history.

Previous History.

Inflammation of lung when ten years old. Enteric fever when seventeen years old. At twenty five years old, when she was pregnant, she fell down and injured her abdomen. This felt sore for one day and patient was quite well again after that. The child was born alive at full term and the labour was easy. Influenza when thirty-one years old. When thirty-two years of age complained of vague pains in the limbs not actually in the joints which were neither swollen, reddened nor painful on movement. No perspiration occurred then. Has suffered from these vague pains on and

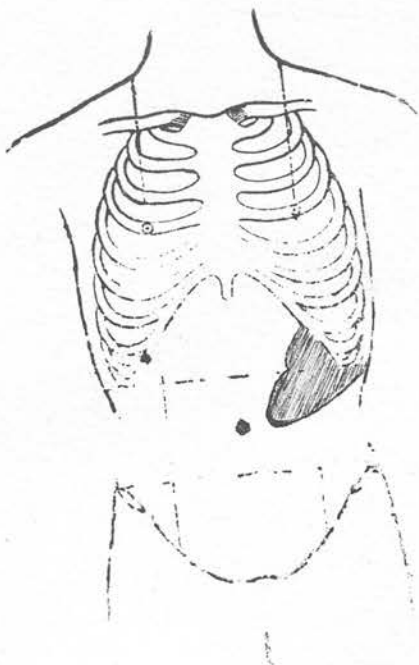
off, but the pains have not necessitated patient to be in bed, until last year when they were so severe that patient had to be in bed for three weeks. Since the last illness the breath has become very short and patient would sometimes faint away. She was diagnosed to be suffering from Rheumatic fever. Denies venereal history.

Present Illness.

Present illness began in March 1916, when a gnawing and continuous pain gradually developed on the anterior and posterior aspects of the left side of the abdomen. The pain did not shoot about and was aggravated by walking and relieved by resting in the recumbent position and also by hot fomentations. She noticed her abdomen was gradually increasing in size. Previous to this illness no increase in size of the abdomen was noticed. Since March her appetite has been failing and she would suffer from a bad taste in her mouth, flatulence and occasionally diarrhoea. she is not certain with regard to her weight but is positive that she is getting thinner.

Shortness of breath on exertion, coughing, with bringing up of thick expectoration, giddiness and fainting are present occasionally. Dropsy has never occurred.

Has had round patches of veins which later burst and cause haemorrhage under the skin of the legs. The size of the patch varies from a sixpenny piece to that of a



Case No 2.



shilling. No other form of haemorrhage has been noticed.

Condition on Examination.

Is not emaciated but the skin of the face and the whole body is pale and has a brownish tinge with freckles dotted here and there. The pigmentation is not darker in the axilla or groin. The mucous membrane is pale. Jaundice is absent. No evidence of enlargement of the lymphatic glands.

Small patches of distended veins are seen on the anterior aspect of both legs. Some of these had evidently burst as haemorrhagic patches are seen amongst the others. The size of the former varies from a sixpence to a shilling and of the latter to a size of half a crown.

Tongue is flabby and moist, and the condition of the teeth are bad. Catamania is regular but the discharge is thin and pale. Temperature is 99° F. Respiration is 22. Pulse is 96 regular in force and rhythm.

The abdomen is distended, the umbilicus is not everted, no veins are visible. It is not rigid but there is tenderness over the enlarged spleen. The spleen is enlarged, hard and smooth. The edge of the organ is sharp, well defined and easily felt. It extends downwards in the mammary line to the level of the umbilicus and forwards and to the right to within an inch to the left of the umbilicus. The notch can be easily felt. The liver is slightly enlarged. Its lower edge extends well below the costal

margin to about one inch in the mammary line. There is no ascites. The spleen is tender, and a bruit is heard over it.

Chest is thin and moves deficiently.

The right lung is resonant all over, but there is deficiency in the entrance of air. No adventitious sounds can be detected. The left lung is resonant all over except at the base, where there is a slight impairment of resonance, and the breath sounds are accompanied by occasional rales.

Heart. There is a diffused pulsation over a wide area of the praecordium.

The apex is in the 6th space in the nipple line.

	Upper border 3rd R.
Right sternal border.	Apex is in 6th space in the nipple line & is $3\frac{3}{4}$ " from mid. line.

A soft blowing systolic murmur is heard all over the praecordia. This does not replace any of the sounds, which are regular in force and rhythm.

#### Nervous System.

Pupils are moderate in size, equal and react to light and accommodation. No optic disc changes can be detected. Abdominal reflexes present.

Knee jerks are present.

No evidence of paresis or paralysis.

Urine. S. G. 1010, acid, shows a deposit of urates, no albumin, sugar or bile can be detected.

Sputum is frothy and viscid contains no tubercle bacillus.

Blood. Wasserman test is negative.

Faeces was examined several times for occult blood but none can be detected.

No evidence of parasites can be detected in the blood. The result of the various blood examinations is given in the appended chart.

The prominent feature of the blood is the presence of a number of abnormal red and white corpuscles, and the intermittency brought about by the effect of arsenic and iron.

The red cells are pale and show slight poikilocytosis and crenation. The cells also vary in size, microcytes and megalocytes being present.

From the time of admittance to the day of the operation, the temperature has been normal as shown in the accompanying chart. During this period pain in the abdomen especially over the spleen, which caused sickness and disturbed sleep occurred occasionally. Cough has also been troublesome but the signs in the lungs were the same as before and the sputum was repeatedly examined, but no tubercle bacilli was found. The telangiectasis and the

haemorrhagic patches persisted throughout. From the date of admission to the day of operation patient has gained  $3\frac{3}{4}$  lbs. in weight and the general health has improved.

The spleen showed no variation in size or consistency in spite of X-ray treatment, and the blood on the whole showed no improvement.

Splenectomy was advised and Mr. Lansdowne removed the spleen on Oct. 4th 1916. Intravenous saline was given at the beginning and right through the operation with the idea of lessening shock. The spleen was removed with no difficulty, there was no adhesion and the pedicle was easily secured. The spleen weighed one pound six ounces.

On the evening of the day after the operation the temperature rose to  $100^{\circ}$  F. and kept swinging for over a fortnight. The wound was examined and found to be clean, and this healed up with no complication. There was consolidation and dry pleurisy of the left base and the pulse and respiration were increased. After this the temperature came down to  $98.2^{\circ}$  F. with an occasional rise to  $101^{\circ}$  F. now and again but the patient showed marked improvement and regained the weight which she lost owing to the operation. Up to the time when patient was discharged the blood condition after the operation showed little or no improvement on the condition before operation.



Patient was readmitted on December 17th 1917 so that a series of blood examinations could be made and also to watch the effect of arsenic and iron on the blood. The result of the blood examination is shown on the appended chart, which shows that on Dec. 20th 1917 when no drugs were administered the red count amounted to 3,500,000, with 40 per cent haemoglobin and 6,500 whites, after a fortnight's administration of arsenic and iron the count was, - red 3,950,000 Hb.. 50% whites 23,750 and on Jan. 29th 1918 the reds had increased to 4,000,000, haemoglobin 60 per cent and whites dropped to 9,000 and on the 6th February 1918 the reds were 4,710,000 Hb.. 60%, whites 7,500. The differential shows the increase in the lymphocytes as mentioned by Dr. Ede.

The patient's general health was excellent and there was no complaints to make. The pigmentation of the skin was still present. No ascites could be detected.

The pathological changes in the spleen is described under the section of pathology.

The above case presents the characteristic symptoms of Splenic Anaemia, and also illustrates the following interesting points:-

1st. The history of vague pains in the limbs, which are probably due to some disturbance in the marrow.

2nd. Telangiectasis.

3rd. The similarity of the blood pictures before

and after operation, with regard to the presence of the abnormal cells, which would strengthen Dr. Michell Clarke's idea that the spleen's eliminative function was diminished; and that the fibrotic changes in the spleen was degenerative in nature.

4th. The variation in the blood brought about by the administration of arsenic and iron before and after operation suggests that the spleen although enlarged was practically functionless.

5th. The beneficial effect of splenectomy.

#### Treatment.

Rest in bed and as much fresh air were ordered.

Diet was as much as the digestive power would permit and consisted of chicken, fish and other nutritious food.

As regards drugs iron and arsenic in the form of:-

Ferri et Ammonii Citratis	gr $\bar{x}$
Liquoris arsenicalis	m $\bar{iii}$ increased to m $\bar{x}$
Aquae	3i

Ter in die, post cibos.

was given and this had to be stopped occasionally owing to gastric disturbance.

X-ray treatment over the spleen was given once weekly. The above measures showed no beneficial effect and splenectomy was resorted to, with a very satisfactory result.

The condition of the mouth was attended to and salol was given for its antiseptic action in the intestine. Alkaline expectorants were also ordered.

Case No.3.

Mrs. Emily Clifford, aged 30, was admitted to the General Hospital, Bristol, under the care of Dr. J. Odery Symes, on May 29th. 1916 complaining of a lump in the left side of the abdomen and jaundice.

Family History.

Father died of heart disease.

Mother died of heart disease.

Two brothers, one is alive and well, and the other died, the cause is unknown.

Husband is alive and well.

Three children alive and well.

Had a miscarriage three years ago when she was six month's pregnant. This was the last time she became pregnant, and it was at this time when the doctor told her of the lump. No tuberculosis in the family history.

Previous History.

Had suffered for some years from vague pains all over the body and also occasional coughing up of sputum streaked with blood. Her doctor said she had neurasthenia. Otherwise has had no other illness or accident.

Denies venereal history.

Present Illness.

The present illness began in July 1913 when her husband noticed that her skin was getting jaundiced, at



that time she was feeling quite well. Her water was not dark, the skin did not itch but her motion was dark in colour. She has been taking Beecham's pills for costiveness for some time.

In November 1913 she noticed that she would get giddy and occasionally felt faint if she did too much.

In February 1914 she had a miscarriage when she was six months pregnant, and her doctor told her that her spleen was enlarged and advised her to seek hospital treatment. She went to a hospital and remained there for two months where they treated her with X-rays twice weekly. She was much improved and the spleen got smaller, but the jaundice still remained. Except for the lump in the abdomen and the slight jaundice, she has been quite well, although her giddiness and faintness still occurred if she did too much.

Three weeks before admittance to the General Hospital, she noticed her appetite was failing and a pain behind the sternum would occur fifteen minutes after every meal. This was relieved by drinking hot water. There was no sickness or vomiting. All the pain disappeared a fortnight after admission and patient insisted on going out. During this period she didn't complain of vague pains or cough. Bleeding from the gums would occasionally occur, but no other form of haemorrhages occurred. On examination she was found to be jaundiced, but not emaciated. The urine contained bile

and the faeces was pale. The spleen was enlarged, hard and not tender, extending downwards to within one and a half inch from the anterior superior spine, and forwards and to the right to within an inch to the left of the umbilicus. The surface was smooth and the sharp edge with the notch was easily felt. The liver was not enlarged and there was no ascites.

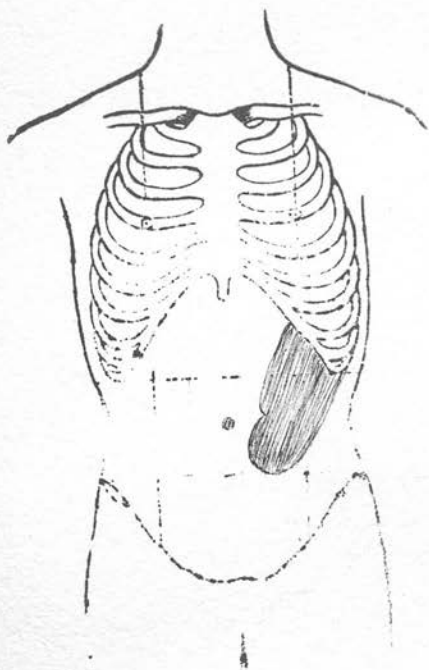
The result of the blood examinations is given in the appended chart.

The prominent feature is the presence of a number of abnormal white cells, but no nucleated reds can be detected either before or after operation; the size of the reds vary and microcytes and megalocytes are present. Slight poikilocytosis and crenation are also present. The effect of iron and arsenic on the blood is also well illustrated in the appended chart. Wasserman test is negative.

Patient was readmitted to the hospital on March 2nd 1917, complaining that the spleen was getting heavier and her abdomen getting bigger; occasional pain over the spleen; weakness and increasing jaundice. Noticed red blood in the motion for two days last Xmas since then her breath has been getting shorter. Her menses has ceased nine months ago.

Condition on Examination.

Patient was not emaciated, but very jaundiced, the sclerotics were also jaundiced. There was no oedema and



Case No 3.

no evidence of enlargement of the lymphatic glands. Teeth were in bad condition. Temperature was  $98^{\circ}$  F. Pulse was 74 regular in force and rhythm. Respiration was 20.

Abdomen was distended, slightly rigid, and tender over the left side. The umbilicus was everted.

The spleen was enlarged, hard and smooth. The sharp edge with the notch could be definitely felt, extending downwards to within an inch from the left anterior superior spine and forwards to the umbilicus, showing a little increase in size since last examined.

The liver was enlarged, the lower border extending one inch below the costal cartilage in the mammary line.

Ascites was also present. These two conditions had developed since she was away.

Chest was well developed and moved freely.

Both lungs were resonant all over except at the bases where there was slight impairment and the breath sounds were accompanied by few rales. Over the resonant part no adventitious sounds were detected.

Heart. The apex beat was not visible but palpable in the 5th space in the nipple line.

	Upper border = 2nd space
Right border was at the sternal margin	Apex beat in 5th space in the nipple line, measuring $4\frac{1}{2}$ " from mid. line.



Soft systolic murmur was heard all over, and the second aortic sound was accentuated.

Sputum showed no tubercle bacilli.

Urine. S. G. 1016, acid, highly coloured, trace of albumin and bile were present. No sugar or blood could be detected.

The blood picture with regards to the presence of abnormal white cells was the same as when she was in before, but the red cells were greatly reduced. No parasites found. Wasserman Test was negative. No occult blood was found in the faeces.

From the time of admittance onwards to the date of the operation the temperature ran a subnormal course with an occasional rise to the normal. During this period patient had two attacks of diarrhoea and complained of occasional pain over the spleen. Slight coughing was also present, but no haemorrhages occurred.

In spite of treatment with X-rays the spleen nor the blood improved and splenectomy was advised. The spleen was removed by Mr. Lansdowne on March 19th. 1917. During the operation the spleen was difficult to remove on account of an accessory attachment and overlapping of the ribs. The spleen weighed two pounds three ounces. The anaesthetic was ether. Not much blood was lost.

The day after the operation, the temperature rose to 101.2° F. and kept high for four days after which it

came down by lysis. The base of the left lung showed signs of consolidation. After this unfortunate occurrence the patient improved and was able to go home on the 20th April 1917.

The blood examinations after the operation was unfortunately not recorded, but when seen some months after the blood showed marked improvement in the red count and haemoglobin and the whites were practically normal but abnormal cells were still present in great numbers. The jaundice was diminishing and patient was bright and said she felt stronger than she had ever been before. No ascites could be detected and the liver was smaller.

#### Treatment.

Rest in bed and as much fresh air as possible were given. The condition of the mouth was attended to by the dental surgeon. Diet consisted of fish, chicken, milk and bread.

Drugs. Iron and arsenic in increasing doses of the liquor arsenicalis were given but had to be stopped twice or three times owing to gastric disturbances.

X-ray treatment once weekly was ordered.

All these measures had no effect so splenectomy was resorted to with, as far as it has gone, a very encouraging result.

The above case presented the characteristic symptoms of Banti's disease. It is interesting from the point of view of the blood picture, for it resembled the blood pictures of cases No.1 and No.2 in having the large number of abnormal cells with the exception of nucleated red cells. The blood condition also showed the variation brought about by the administration of iron and arsenic which was also present in Cases No.1 and 2.

The similarity of the blood pictures before and after operation, with regards to the presence of abnormal cells is also shown in this case.

This case resembled case No.2 in having symptoms of vague pains which might have been due to disturbance of bone marrow.

It shows the beneficial effect of Splenectomy.

Case No.4.

Mrs. Edith Stenner, aged 25, was admitted to the General Hospital, Bristol, under the care of Dr. Michell Clarke, on November 30th 1915, complaining of occasional pain around the umbilicus and a lump in the left side of the abdomen.

Family History.

Father died of heart failure.

Mother is alive and well.

One brother is alive and well.

Two sisters are alive and well.

Husband is alive and well.

Two children are alive and well.

Never had a miscarriage.

No Tuberculosis in the family.

Previous History.

In 1905 had appendicitis and was operated on.

In 1906 had a cough and was sent to Winsley Sanatorium.

In 1909 was again sent to Winsley Sanatorium and discharged as cured.

In 1914 contracted malarial fever in China.

Was in Malta from 1911 to 1913, and had no illness there.

She did not drink milk whilst in Malta.

Denies venereal history.

Present Illness.

In August 1915 patient noticed a hard lump in the

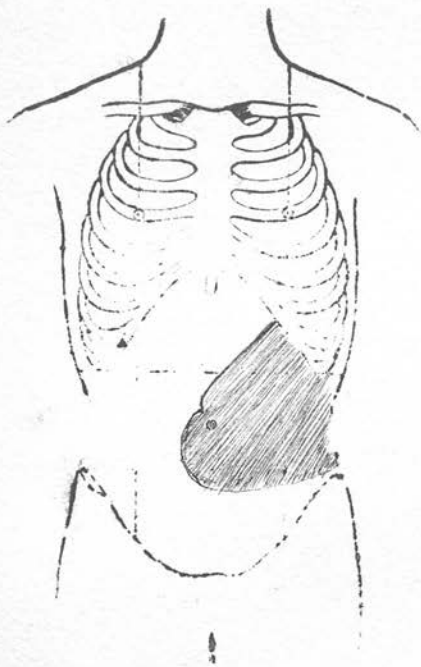


left side of her abdomen, which she was able to move about easily without causing pain. Since then it has been getting larger and now it is immovable. At the same time she has noticed her abdomen to be getting larger and lately pain and tenderness in the abdomen especially over an area above and to the left of the umbilicus, have developed.

Ever since she has been in China she has had several attacks of fever with rigor and sweating which lasted from four to six hours. She used to suffer from this every other day for the first two months after return from China, but now only once a week. The last attack was on November 28th 1915. All this time she has been taking five grains of quinine three times daily. Apart from the ague she suffers from occasional night sweating. Has never had epistaxis nor any form of haemorrhage. She complains of shortness of breath, palpitation and discomfort around the heart on the slightest exertion. She has a constant cough with little viscid expectoration, this is worse at nights. Has lost weight and getting thinner these last few months. Has never been jaundiced, or noticed any oedema.

#### Condition on Examination.

Patient is thin. The face and the whole body is earthen in colour and anaemic. No pigmentation can be detected. The mucous membrane is pale, no icterus can be seen in the conjunctivae. The tongue is moist, slightly furred and tremulous. The teeth are in a bad condition.



Case No 4

There is no evidence of enlargement of the lymphatic glands. Skin is moist and patient has a hacking cough, with no expectoration to speak of. No oedema is present. Temperature is 101.2°F., Respiration is 30 and Pulse is 116 weak but regular in force and rhythm.

Abdomen is distended, not rigid or tender, and moves as a whole on respiration. The umbilicus is everted. An old appendix scar is present.

The spleen is greatly enlarged, hard, smooth and moves slightly with respiration. It is tender to touch. It presents a sharp, well defined and easily felt edge with a definite notch. The organ extends downwards to just above the pubis and forward and to the right to well beyond the middle line.

The liver is not enlarged and no fluid can be detected.

Chest. Thin but moves equally and freely on respiration. The left lung is resonant all over, the breath sound is vesicular in type. Vocal resonance and fremitus are normal. No adventitious sounds can be heard.

The right lung is resonant all over except in the third interspace anteriorly where the resonance is slightly impaired and a few clicks can be heard, otherwise the breath sound, vocal resonance and fremitus are normal and no adventitious sounds can be detected elsewhere.

Heart. There is a diffuse pulsation in the 5th and 6th spaces. The apex beat is felt in the 6th space one inch external to the mammary line.

There is venous pulsation in the neck and no epigastric pulsation is noted.

	Upper border = 3rd R.
$\frac{1}{4}$ " from right sternal border.	$4\frac{1}{4}$ " from med. line.

Faint systolic murmur is heard all over the heart.

Nervous System.

Pupils equal and react to light and accommodation.

Abdominal reflexes present.

Knee Jerks present.

No signs of paralysis are present.

Sputum - shows no tubercle bacilli.

Urine. S.G. 1019, acid, shows a deposit of urates, a trace of albumin is present, but no sugar, blood or bile.

Blood. Wasserman Test is negative.

No malarial parasites can be detected on several examinations.

The result of the blood examinations is given in the appended chart.

The blood shows a diminution in the red and white



cells and also in the haemoglobin. The differential counts shows an increase in the lymphocytes, and no abnormal cells. The condition of the blood remains unaffected although iron and arsenic were given.

From the time of admittance onwards the temperature chart shows an irregular pyrexia. The rise of temperature occurs usually daily but not at any definite time.

During the period she was in hospital she had no rigor, but suffered from a hacking cough and profuse night sweats.

In spite of the administration of iron and arsenic and the X-ray treatment neither the spleen nor the blood improved, and as her general condition was getting worse and she was losing weight, it was decided to send her to the convalescent home before advising for surgical interference. She left on February 8th 1916 and returned to the hospital on March 1st 1916. Her condition if anything was worse than before. Except for oedema of the ankles the symptoms and signs were as before. She requested to be sent home, and as her condition contra-indicated surgical interference, her request was granted. She died two months later.

#### Treatment.

Rest in bed and as much fresh air as possible were given. The condition of the teeth was attended to by the

dental surgeon.

Diet was plentiful and nutrituous and consisted of milk, fish, chicken etc.

Iron and arsenic in the form of -

Ferri et Ammonii Citrates gr  $\bar{x}$

Liquoris Arsenicalis m  $\overline{iii}$  - m  $\bar{x}$

Aquae 3i

Ter in die, post cibos.

were given.

Quinine Sulphate gr  $\bar{v}$  was given three times daily but had no effect on the temperature.

X-ray treatment once weekly over the spleen was ordered, but produced no effect.

The above case is of interest in many respects.

1st. Owing to the leucopenia and the splenomegaly the diagnosis of splenic anaemia was entertained but this diagnosis was made improbable not only by the previous history of pulmonary trouble which necessitated her being removed to Winsley Sanatorium but also by the irregular pyrexia, night sweats, coughing and the sign in the right lung. The enormous splenomegaly was probably due to tuberculosis with malaria superadded. It was unfortunate that autopsy could not be obtained but from the

above reasons the diagnosis of splenic anaemia or Banti's disease may be eliminated. This being so it would seem that the presence of abnormal red and white cells are essential in splenic anaemia or Banti's disease, as shown in the blood examinations of the other cases.

Case No. 5.

Mrs Elizabeth Faux, thirty-six years of age, was admitted to the General Hospital, Bristol, under the care of Dr. J. Odery Symes, on Thursday 25th, 1914, complaining of weakness and breathlessness.

Family History.

Husband is alive and well.

Four children are alive and well, the youngest is three months old. Never had a miscarriage. All the labours have been easy. There is no consumption in the family.

Previous Illness or Accident.

Has never been abroad.

Had Enteric when twenty years of age.

Had frequent bleeding from the nose some years ago.

Had all the teeth from the upper jaw removed ten years ago, as they were very bad.

Never had an accident or blow on the abdomen.

Present Illness.

Has been quite well until the birth of her last child, three months ago. Since then never picked up strength and has grown progressively weaker. The breath has also become very short on the slightest exertion. During the last few weeks the ankles have become swollen, but not reddened nor painful.



Later the swelling travelled up the legs, abdomen and face, with this a troublesome cough developed. Never noticed difficulty in fastening the waist band of her skirt until the swelling affected her abdomen. Except for the bleeding from the nose which occurred some years ago and none lately, never noticed any bleeding under the skin, nor vomited or passed blood or dark matter by the bowels.

Condition on Examination.

The face and the whole body are pale and slightly oedematous; a distinct lumbar pad is present. There is a malar flush on the cheeks. No discoloration of the skin can be detected. The mucous membrane is pale. There is slight icterus in the conjunctivae which are fatter than normal. The teeth of the lower jaw are ill kept. There is no enlargement of the lymphatic glands or tonsils. Abdomen is distended, slightly oedematous and skin is shiny. The umbilicus is not everted. No superficial veins are congested.

The spleen is greatly enlarged, hard and not tender. The surface is smooth and its edge is sharp and presents the notch. It moves slightly with respiration. Vertically it measures seven inches from the costal margin in the anterior axillary line and its edge is three inches and a half from the middle line at the level of umbilicus.

The liver is slightly enlarged. Its upper border is at the level of the 5th rib. The lower edge is distinctly felt to be hard, round, slightly tender, especially in the nipple line, but not nodular. There is little fluid in the abdomen. Kidneys are not palpable.

#### Heart.

The apex beat is difficult to feel. It is diffused over the 4th and 5th spaces and extends outwards to the left nipple line.

	3rd Rib.
Right Sternal	In 5th space
Border	in nipple line.

Heart sounds are weak, rapid but regular in time.

Faint systolic murmur is heard all over the heart, but loudest over the pulmonary area.

#### Lungs.

The bases of both lungs are slightly dull. The breath sounds are harsh with prolonged expiration and few rales. Over the rest of both lungs they are harsh but there are no accompaniments. Cough is slight and there is a small amount of frothy sputum which on examination shows no tubercle bacilli.

Pupils. Are equal and react to light and accommodation; no optic changes can be detected on ophthalmoscopic examination.

Urine. S. G. 1015, strongly acid, shows a deposit of urates.

There is a trace of albumin, but no sugar, no blood or bile can be detected.

Blood. Shows no parasites. Blood counts etc., see appended chart.

The prominent feature of the blood is the presence of a number of abnormal red and white corpuscles.

The red cells are paler than usual and crenated.

Slight poikilocytosis, punctate basophilia, chromatophilia are present.

An interesting fact in the case is that in the last blood examination the haemoglobin is above normal.

From the time of admission onwards the temperature chart showed an irregular pyrexia of a remittent type.

The pulse was of moderate volume and rapid but later improved.

The respiration rate never exceeded twenty five.

The general health improved, and although the red blood count and haemoglobin improved yet leucopenia and the abnormal cells remained the same. After four exposures to the X-rays the spleen showed a little diminution in size. The general oedema showed diminution, but the ascites remained. The icterus of the conjunctiva was deeper and bile in the urine noticed for the first time on April 1st, 1914.

Pain was occasionally present over the spleen.

Diarrhoea was occasionally present.

Iron & arsenic in the form of Ferri et Ammonii Citratis gr. x.

Liquoris Arsenicalis m iii - mxv.

Aquae.

3i

Ter in die, post cibos.

were given from the beginning.

X-ray treatment twice weekly was ordered on March 6th, 1914. B naphthol tablets gr. v. tds was ordered on March 30th, 1914, with the hope of arresting the diarrhoea.

Splenectomy was advised but refused and patient insisted on leaving the hospital on April 2nd 1914.

The last heard of this patient was that she died three months after leaving the hospital.

This case presents the characteristic symptoms of Banti's disease.

It illustrates as in the other cases the presence of the large number of abnormal red and white cells, and The progressive and fatal course of the disease without splenectomy.



THESIS  
on  
SPLENIC ANAEMIA AND BANTI'S DISEASE.

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Albert Liat Juay Lim,  
M. B., Ch. B., D. T. M.,

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March 30th, 1918.

Blood Examination Charts.  
Temperature Charts.  
Blood Pictures.

of  
Cases under observation.  
for  
Thesis  
on

Splenic Anaemia and Banti's Disease.

March 30th, 1918.

Albert Liat Quay Lim.

M.B., Ch. B., D. T. M.

# Case No. 1 Alexander Rawlings Results of Blood Examinations

Results of Blood Examinations													
Date	Red Cell Count per c. mm.	Total Leucocytes per c. mm.	Haemo-globin per cent.	Haemo-nucleated Red Cells counted per 300 Leucocytes	Differential Leucocyte Count							Remarks	
					Finely granular polymorpho-nuclears	Small lymphocytes	Large lymphocytes	Mononuclears	Baso-philic	Leuco-blasts	Giantly granular eosino-philic		Myelo-cytes
1916													
Mar. 27	3,200,000	6,400	55%	3	60	10.5	4.5	17	3	0	1	4	Red cells show they are slightly pale. Slight polycythemia & active mitosis in the nuclei of many cells are present. Size of cells vary. On 27.3.16 Lq. Arsenic also m <sup>ij</sup> was ordered tid. & was increased to m <sup>v</sup> on the 6.4.16. This was stopped on the 12.4.16 on 14.5.16 arsenic was resumed. On 26.5.16 was discharged as out-patient and to take medicine & carry out x-ray once weekly. X-ray was ordered on the 14.5. April 1916 Was readmitted & the amount of arsenic was reduced from m <sup>v</sup> to m <sup>ij</sup> on 1.9.16. This was increased by m <sup>ij</sup> weekly. On 4.10.16 Temp. 101.4°F " 5.10.16 " 102°F " 6.10.16 " 100.2°F Patient complained of pain over spleen which was harder & larger. During the operation there was a good deal of haemorrhage. On 18.10.16 patient was comfortable & Temp. was normal. On the 19.10.16 Temp. showed a slight
Apr. 7	4,002,000	10,000	70%	10.5	54.7	16.5	8.2	16.7	3.5	5.5	.2	10.2	
Apr. 14	3,944,000	6,100	60%	5.2	52	11.3	8	20.6	2.6	.8	.6	6.6	
May 9	5,190,000	12,400	75%	14.3	29.2	14.5	20	27.2	0	1.5	1.6	6.9	
Sept. 1	5,620,000	15,620	75%	8	54	10	16	10	2	0	2	6	
Sept. 20	3,570,000	34,37	40%	8	50	20	10	8	2	0	2	8	
Oct. 9	4,500,000	10,000	60%	14	52.8	14.4	10.4	22.4	0	1	.4	2	
Oct. 16													Splenectomy performed
Oct. 18	2,900,000	10,312	63%	8	84	1	2.3	12	1	0	0	1	
Oct. 23	2,600,000	21,500	70%	10	80	3	3	12	.3	0	0	2	

Splenectomy performed

Red cells show they are slightly pale. Slight polycythemia + active mitosis in the nuclei of many cells are present. Size of cells vary. On 27:3:16 Lig. Arsenically m<sup>ij</sup> was ordered tid. + was increased to m<sup>v</sup> on the 6:4:16. This was stopped on the 12:4:16 on 4:5:16 arsenic was resumed. On 26:5:16 was discharged as out-patient and to take medicine + carry out x-ray once weekly. X-ray was ordered on the 14:5:16 April 1916 Was readmitted + the amount of arsenic was reduced from m<sup>v</sup> to m<sup>ij</sup> on 1:9:16. This was increased by m<sup>ij</sup> weekly. On 4:10:16 Temp. 101.4°F " 5:10:16 " 102.0°F " 6:10:16 " 100.2°F Patient complained of pain over spleen which was hard + larger. During the operation there was a good deal of haemorrhage. On 18:10:16 patient was comfortable + temp. was normal. On the 19:10:16 temp. showed a slight

Case No. 1 - continued -

Date.	Red cell Count per c. mm.	Total Leucocytes per c. mm.	Hemo- globin.	Ruled Red Count per 300 Leucocytes	Differential Leucocyte Count.						Remarks.
					Small lymphocytes - nuclei	Large lymphocytes	Transit lymphocytes	Basophils	Granulocytes	Myelo- cytes	
1916											
Nov. 1	3,600,000	15,750	50%	13	90	23	2	19	3	0	1
Nov. 4	3,800,000	25,000	50%	5	79	1	6	13	0	0	1
Nov. 11	3,000,000	19,000	40%	52	92	16	26	33	0	0	0
Nov. 20	2,000,000	17,160	35%	2	81	1	6	14	5	0	0
Nov. 26											
Nov. 29	4,790,000	15,800	50%	0	72	6	92	105	15	0	0
1917											
Oct. 7	6,630,000	19,159	75%	95	525	115	13	85	15	45	7
1918											
Feb. 4	5,700,000	16,875	85%	84	545	135	145	84	28	0	3

Haemoglobin estimated by Gowen's apparatus.

rise of temperature & the following four weeks the temperature was hectic - rising in the evening to about 101° to 102° F. During which antistreptococcal serum was administered. The blood showed a polymorphonuclear leucocytosis. On the 23:10:16 Seric St. Ammonia Cit. 97 x 34 Ag. ac 100 p.c. was ordered & stopped on 1:11:16 On Nov. 26: Resection of rib was performed & henceforth patient showed marked general improvement. The blood also improved. Seen on 7:10:17 - a year after splenectomy patient looked well but still complained of breathlessness on exertion. When seen on Feb. 4, 1918 patient was looking very well & said he was stronger than ever he was. He was working 16 hours a day.



Case No. 2, Annie Long

Case No. 2, Annie Long												
Date.	Red cell count per c. mm.	Total Leucocytes per c. mm.	Hæmo-globin	Nucleated Reds counted per 300 leucocytes.	Differential Leucocyte Count.						Remarks.	
					Small Lymphocytes	Large Lymphocytes	Transi-tionals	Basophils	Slightly granular eosinophilic	Myelo-cytes		
												Finely granular polymorpho-nuclears
1916												
Aug. 14	3,050,000	3,100	50%	4	74	10.5	5.5	6	0	2	2	Red cells are pale + show slight crenation and poikilocytosis.
Aug. 28	3,810,000	11,875	50%	6	76	2	11	6	1	2	2	Microcytes and megakaryocytes are present.
Sept. 9	3,060,000	4,530	50%	4	78	10	0.75	2.6	0	2	14	On 16:8:16 Ferri-d Ammonii Cit. gr. $\bar{x}$ dig. Arsenic acid m. $\bar{m}$ Aquae sola p.c. was ordered.
Sept. 13	3,400,000	5,312	50%									Arsenic was increased to m.vt.
Sept. 27	4,540,000	14,062	60%	3	65.3	13.6	8	10	0	3	0	On 20:8:16 On 18:8:16 X-ray once weekly was ordered.
Oct. 4												On 14:9:16 Medicine was discontinued but resumed again on the 14:9:16.
Oct. 7	3,000,000	11,600	55%	3	80	2	3	10	2	0	4	No hæmorrhage, no cough no rise of temperature.
Oct. 17	3,100,000	10,000	50%	5	59	6.7	7.2	22.2	2.2	1.5	2	Operation was not accompanied by much loss of blood.
Oct. 25	3,210,000	12,500	50%	3	57	22	12	7	0.25	3	0	On 5:10:16 temperature rose to 100° + kept swinging for five weeks. definite signs of consolidation detected in the lungs.
Oct. 27	3,006,000	10,000	50%	7	90	1	3	3	0.3	0	2	The same mixture of iron and arsenic was given again for first time after the operation
Nov. 11	3,000,000	12,000	50%	1	56.6	5.3	22	1	1	2.6	3	on 17:10:16 + was discontinued on 30:10:16 + resumed on 17:11:16
Nov. 14	3,900,000	9,375	55%									
Nov. 29	4,130,000	18,125	60%	1	43	29	9	9	3	4	2	

Splenectomy Performed

Case No. 2 - continued -

Date.	Red cell count per c. mm.	Total Leucocytes per c. mm.	Hemo-globin	Nucleated Reds counted per 500 Leucocytes	Differential Leucocyte Count.						Remarks
					Small lymphocytes	Large lymphocytes	Transi-torials	Basophils	Granulo-phils	Myelo-cytes	
1916											
Dec. 15	3,860,000	12,500	55%	2	48	9	8	2.4	2.4	2	
Dec. 27	3,860,000	7,000	50%	3	50	10	10	2	3	2	
1917											
Dec. 15	3,700,000	10,000	50%	3	46	19	10	2	4	8	When seen on 15:13:17 over one year after the operation, patient was quite well & felt very much stronger than she has ever been before but got short of breath on exertion.
Dec. 20	3,500,000	6,500	40%	3	45	20	15	2	3	3	On 20:12:17 Ferri et Anemonie Ct. gr. x Liq. Arsenicalis m. iii Aqua Ferri t. d. s p.c. Vitel. Ferri t. d. s p.c. were ordered.
1918											
Jan. 7	3,950,000	23,750	50%	4	50	20	10	2	2	6	When discharged on 19:2:18 patient felt stronger & didn't get short of breath on exertion.
Jan. 29	4,000,000	9,000	60%	4	30	24	15	1	7	6	
Feb. 6	4,710,000	7,500	60%	3	33	29	14	2	4	5	

Case No. 4 Edith Stenger Results of Blood Examinations

Date.	Red cell count per c. mm.	Total Leucocytes per c. mm.	Hemo- globin	Differential Leucocyte Count.			Remarks.
				Finely granular Polymorpho- nuclears	Small Lymphocytes	Large Lymphocytes	
1915 Dec. 1	3,960,000	4,375	50%	45	45	10	Red cells are pale & crenated. Slight poikilo- cytosis is present. Lymphocytosis is the constant blood picture. During her stay the temperature shows an irregular pyrexia. There was no rigor but a constant hacking cough.
Dec. 6	3,900,000	4,300	55%				
Dec. 10 1916	3,850,000	3,000	55%	35	55	10	
Jan. 10	3,830,000	3,000	50%	45	40	15	On 8:12:15 Fever et Ammonii Ct. gr X Legionis Arsenicalis m III Agras t d s p.c.
Jan. 17	3,830,000	2,500	58%				was ordered, & the arsenic was gradually increased to m X.
Jan. 24	3,800,000	2,200	50%				On 29:12:15 X-ray once weekly was applied over the spleen but neither of the above treatment improved either the blood or the general condition.
Mar. 8	3,000,000	1,250	50%	40	50	10	No parasite was found in the blood. Splenic puncture revealed no Leishman Donovan's bodies. Sputum showed no tubercle.

Case No 3 Emily Clifford Results of Blood Examinations

Date.	Red Cell Count per c. mm.	Total Leucocytes per c. mm.	Hemo-globin	Leucocyte Count.						Remarks.
				Small lymphocytes	Large lymphocytes	Transi-tionals	Baso-phils	Coarsely granular basino-phils	Myelo-cytes	
1916										Red cells are pale + show slight poikylotosis, variation in size. Microcytes and megakaryocytes are present.
May 29	3,330,000	4,300	50%							On 30.5.16 Sig. Arsenicosis m <sup>iii</sup> tdx.
June 11	4,110,000	11,000	60%	21	22	7	1	0	4	was ordered + the dose was increased to m x on 10.6.16 but had to be discontinued on 12.6.16 + was discharged.
June 16	4,060,000	9,600	60%	6	11	1	5	2	5	Little loss of blood during operation.
Mar. 18				Splenectomy performed.						On 19.3.17 the temperature rose to above 100°F + bronchitis was detected for the following six days.
Mar. 19	2,960,000	12,200	40%	20	18	10	4	3		On 1.4.17 Arsenic was again given + patient was discharged on 2.4.17
Apr. 5	4,100,000	22,776	60%	16	4					was seen in June 15 <sup>th</sup> 1917 + looked well, but colour was still bad. Was put on Arsenic again.
June 29	6,130,000	18,750	70%	26.6	13.3	2.5	6	3.1	.8	Seen for last time in Nov. + looked well + patient had no complaint to make.
Nov. 27	5,300,000	9,850	65%	19	16	10	4	3	2	

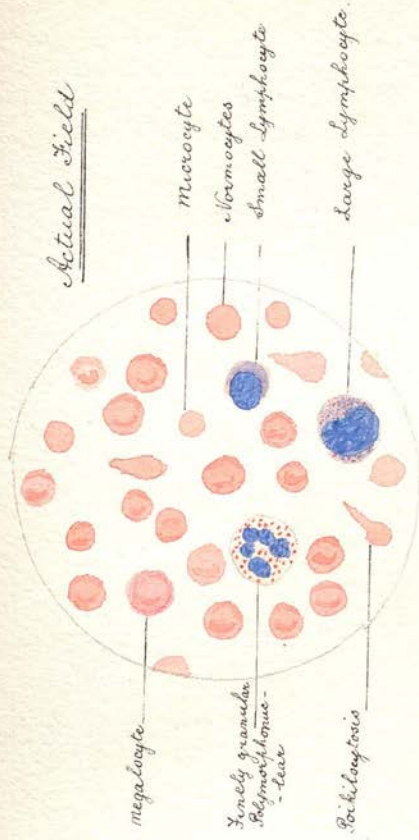


# Case No. 5 Elizabeth Faux. Results of Blood Examinations

Date	Red Cell Count per c. mm.	Total Leucocytes per c. mm.	Haemoglobin-globin	Nucleated Red Cells counted per 100 leucocytes	Differential Leucocyte Count.						Remarks
					Small lympho-cytes	Large lympho-cytes	Transi-tionals	Basophils	Eosinophils	Myelo-cytes	
1914											Admitted on Feb. 25 <sup>th</sup> 1914, with temp. 101.8 F pulse 120, resp. 21
Feb. 27	2,870,000	7,010	55%	10	60	35	1	1	2	1	On 26: 2: 14 Fever et Anemia C.T. 91 x Lg. Arsenicals m <sup>iii</sup> Agave 37 tubo. p.c. was ordered. On 2: 3: 14 Temp. 98.2 F, resp. 20, pulse 102. No haemorrhage, nor pain complained of. On 3: 3: 14 Lg. Arsenicals was increased to m <sup>v</sup> and on 6: 3: 14 X-ray treatment twice weekly commenced. On 9: 3: 14 Lg. Arsenicals was increased to m <sup>xv</sup> .
Mar. 2	2,900,000	7,200	54%	16	54	41	4	1			The red cells were paler than usual + crenated. Slight poikilocytosis, punctate basophils + chromatophils were present.
Mar. 10	3,750,000	7,200	73%	14	59	29	2	2	3	2	On 20: 3: 14 The C.I. was about 1. On the above treatment the blood improved but the splenomegaly and the general health showed no improvement.
Mar. 20	4,050,000	8,800	93%	39	50	44	3	2	1		

Haemoglobin was estimated by Fowers apparatus.

Blood Picture before Splenectomy.



1/2 Oil Immersion



Leishman's stain.



Actual Field

Case No 1.

Blood Picture after Splenectomy.

1/2 Oil Immersion.

Leukemia's stain



[illegible]

For the remaining five weeks the temperature chart is practically identical. Before leaving hospital on May 6th, 1916, patient's weight was 11 st. 3½ lb.

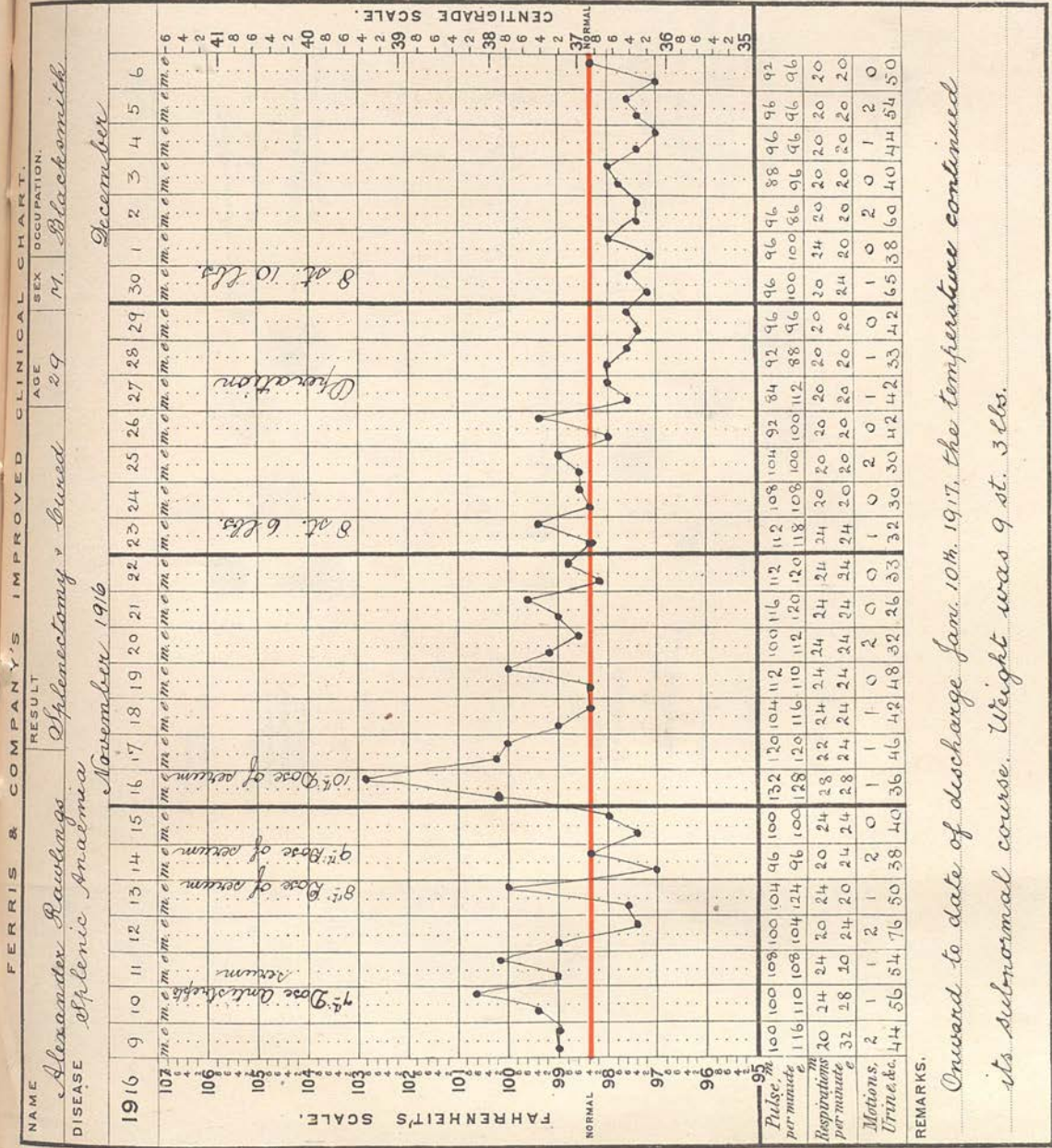


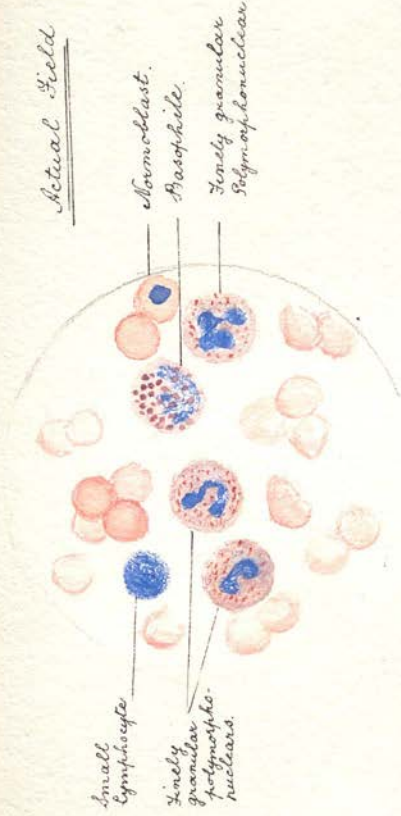
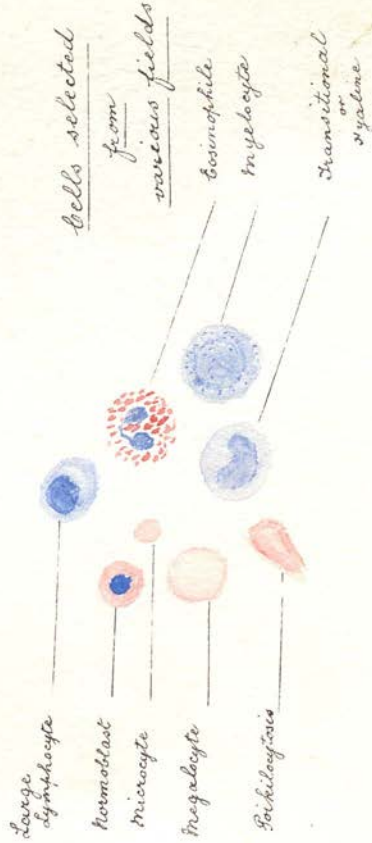
[illegible]

From the date of admission Aug. 31 st. 1916 to Oct. 12th. 1916 the temperature ran a subnormal course similar to the previous chart. Weight was 10 st. 9 1/2 lbs.

WHOLESALE & EXPORT DRUGGISTS, SURGICAL INSTRUMENT MAKERS &c. BRISTOL.





Blood Picture before splenectomy1/12 Oil ImmersionLeishman's stain



NAME	RESULT	AGE	SEX	OCCUPATION																									
Annie Long	Splenectomy + cured	36	F	Housewife																									
DISEASE	Splenic Anaemia																												
August, 1916																													
1916	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	6	7	8	9	10	
107	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	m.e.m.	
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Pulse, per minute	96	100	92	88	80	92	92	80	88	84	88	84	80	94	88	84	88	80	84	84	80	84	80	80	84	88	80	88	84
Respirations per minute	20	18	20	20	20	18	20	20	20	20	15	22	20	20	20	20	20	22	20	20	20	20	18	20	20	24	20	20	20
Motions, Urine, etc.	2	1	1	1	1	4	4	1	3	0	4	1	4	2	3	2	2	1	4	2	2	1	2	2	0	2	2	2	3

Fahrenheit's Scale.

Centigrade Scale.

Normal

Normal

Admitted

8 wt. 7 lbs.

8 wt. 4 1/2 lbs.

8 wt. 6 1/2 lbs.

8 wt. 9 1/4 lbs.

September

REMARKS.



NAME

RESULT

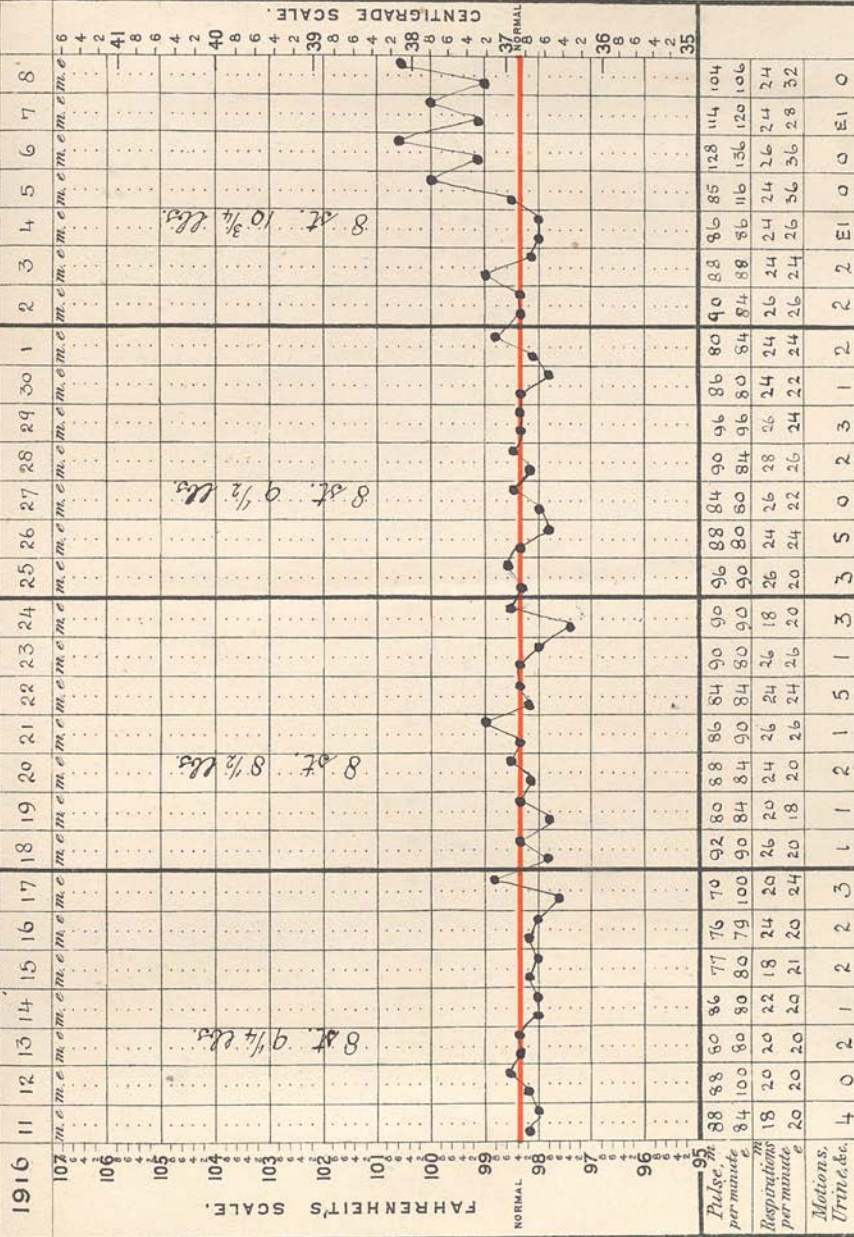
AGE

SEX OCCUPATION.

Annie Long  
Splemic Anaemia

September 1916

October



REMARKS.

Operation on October 4th. 1916.

NAME

RESULT

AGE

SEX

OCCUPATION

Annie Long Anæmia  
splenic

October 1916

November

DISEASE

1916

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NAME

RESULT

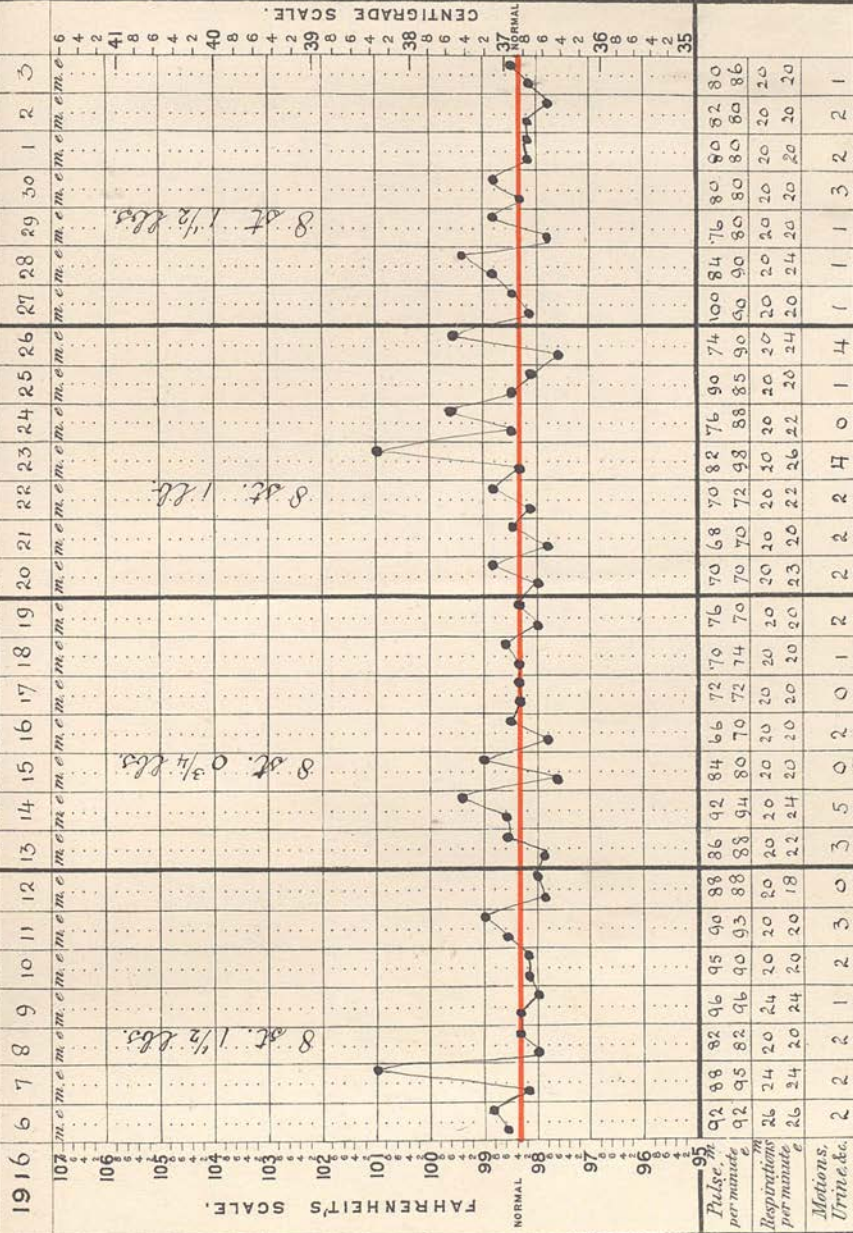
AGE

SEX

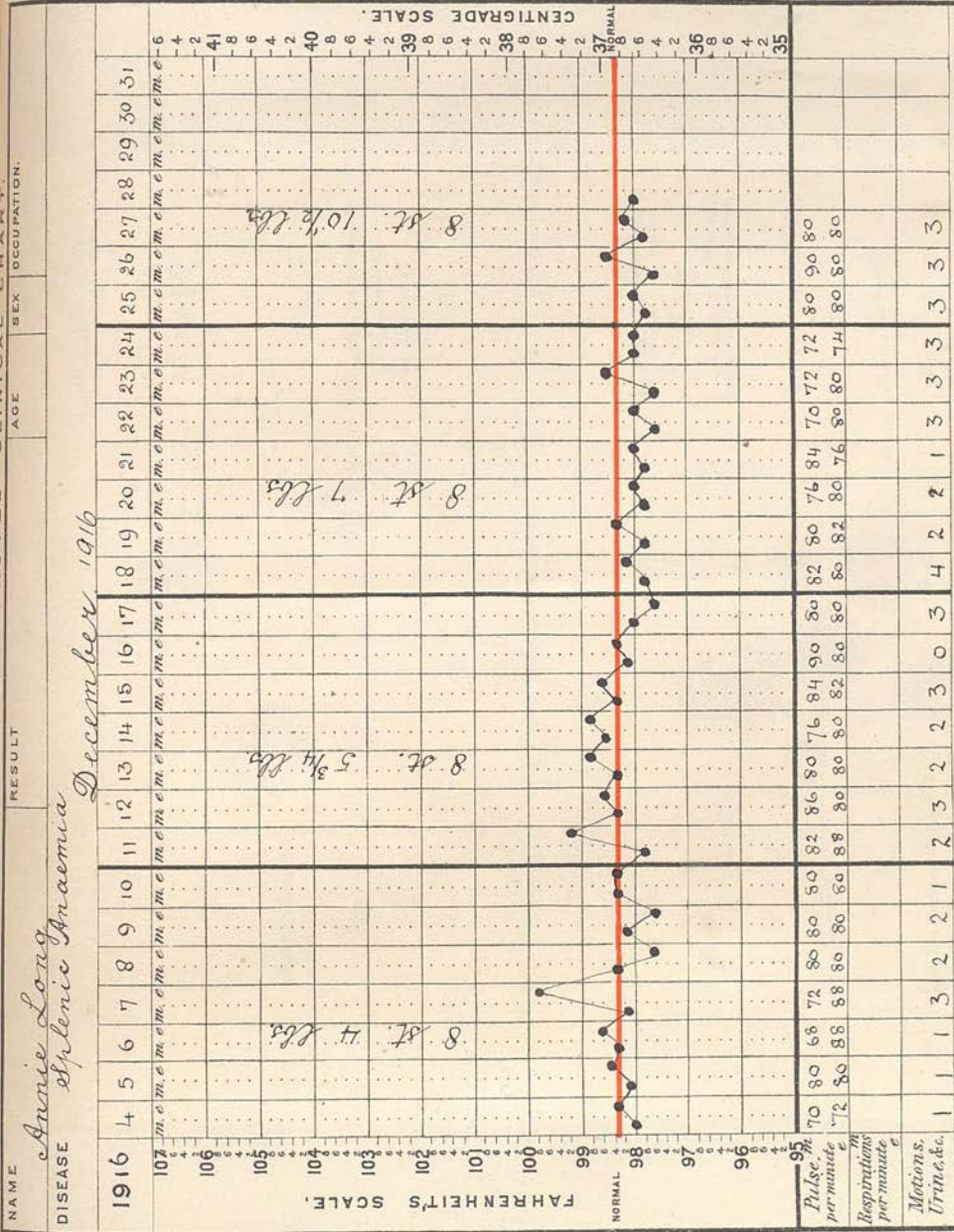
OCCUPATION

DISEASE *Annie Long  
Splemic Anaemia*

*November, 1916*



REMARKS.

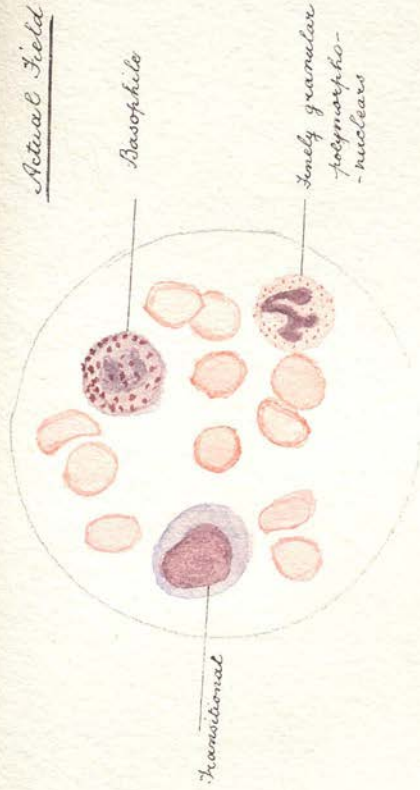


REMARKS.

Respiration rate was between 20 + 22 during last three weeks.

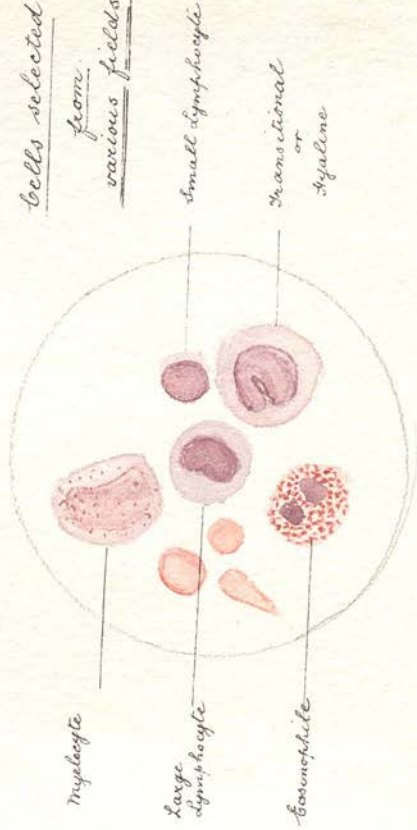


Actual Field



1/12 Oil immersion

Cells selected from various fields



Leishman's stain







NAME

Emily Clifford

RESULT

Splenectomy + Cured

AGE

30

SEX

F

OCCUPATION

House wife

DISEASE

Bant's Disease.

April 1917

1917

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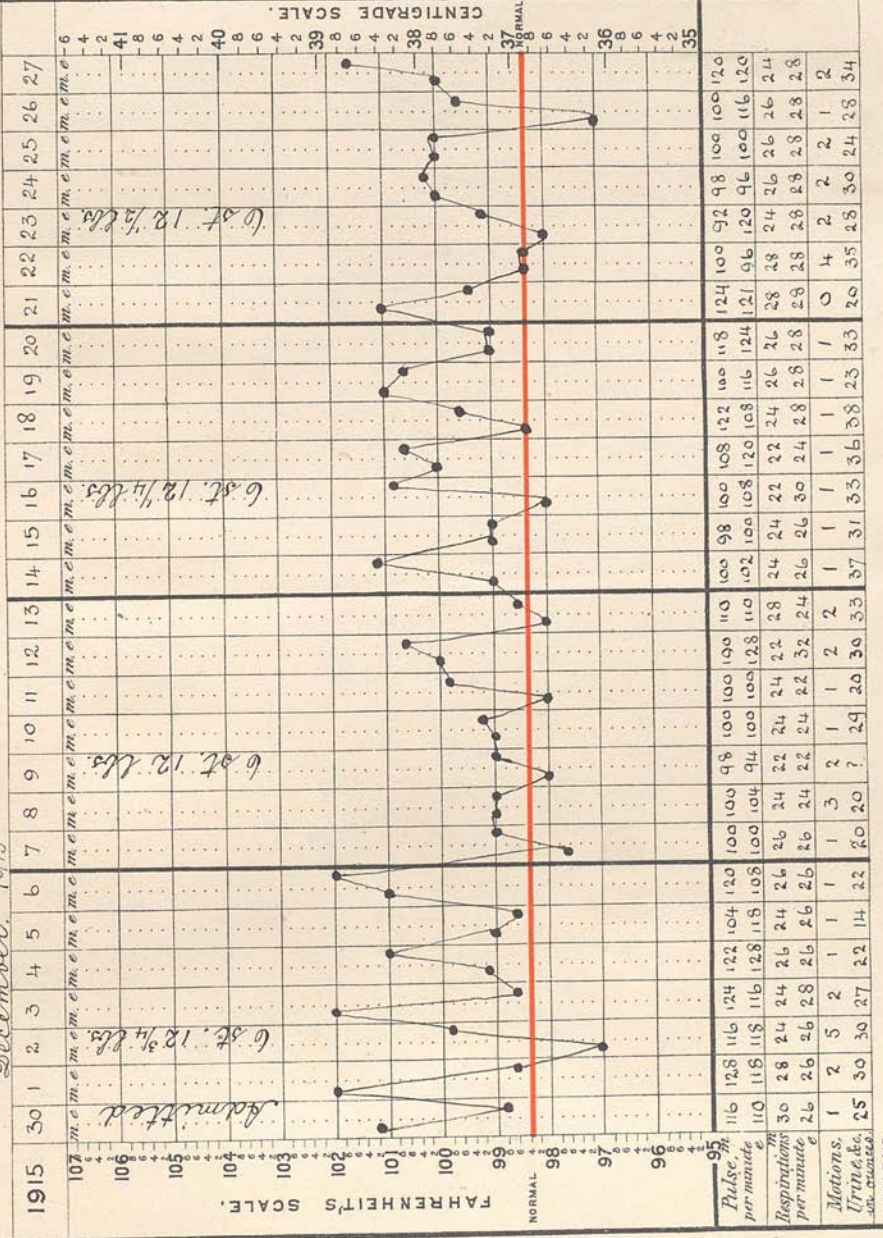
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NAME Edith Stenner. DISEASE Splenic Anaemia. RESULT Died after leaving hospital AGE 25 SEX F OCCUPATION Housewife.

December, 1915



The diagnosis is probably Tuberculosis with malaria.





January

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1. **Introduction**


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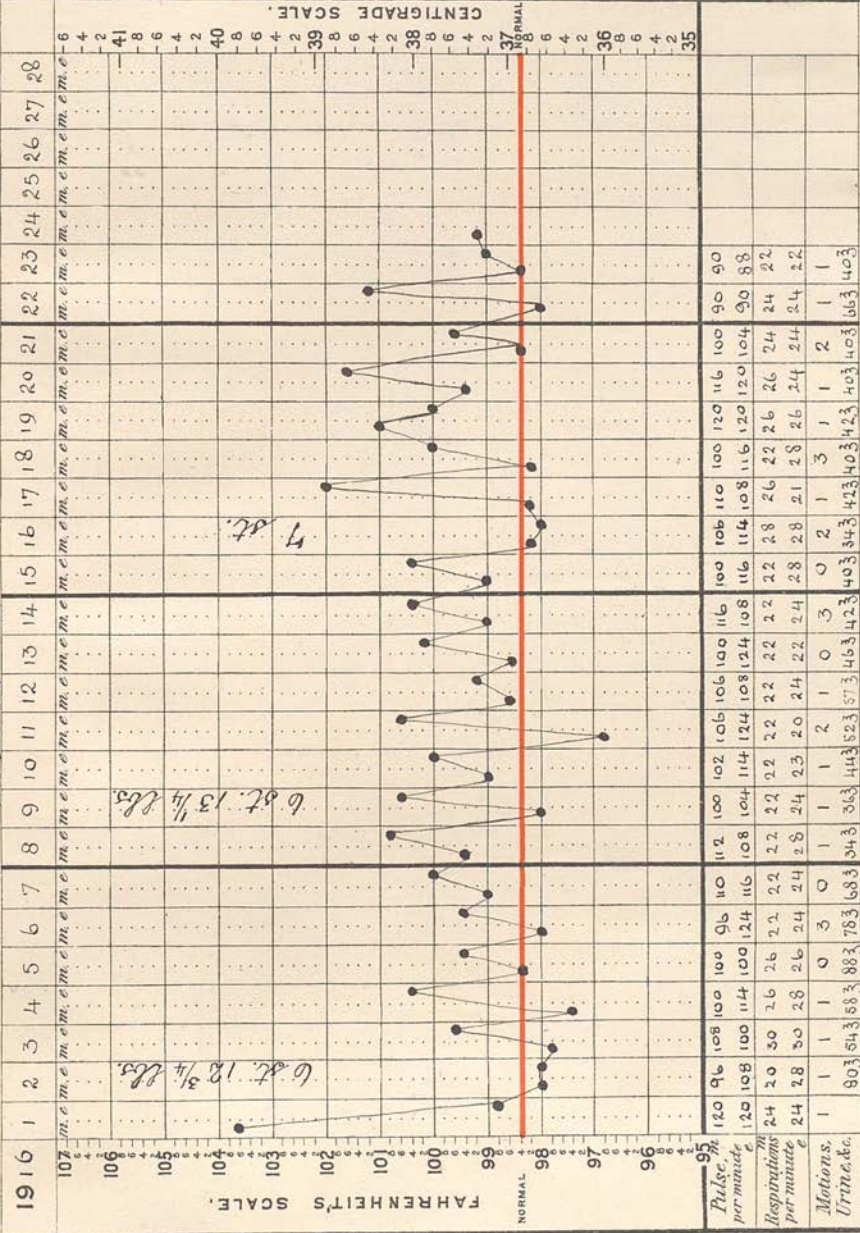
Ferris & Co. Ltd Bristol

NAME

Edith Stenner

DISEASE? Splenic Anaemia

March 1916



Patient was readmitted.

The diagnosis is probably Tuberculosis with malaria

WHOLESALE & EXPORT DRUGGISTS, SURGICAL INSTRUMENT MAKERS & C. BRISTOL.



NAME	RESULT	AGE	SEX	OCCUPATION
Elizabeth Foxe	Died 2 months after leaving hospital	34	F	House wife
DISEASE	Scarlet Fever	February	March	1914

DATE	TEMP.	PULSE	RESPIRATIONS	MOTIONS	URINE	REMARKS
1914 25	102	120	21	2	?	Admitted
26	102	108	20	3	1043	
27	102	100	18	1	157	
28	102	108	18	1	213	
1	102	106	18	2	213	
2	102	100	18	3	193	
3	102	120	20	2	243	
4	102	96	16	2	333	
5	102	90	16	2	333	
6	102	92	20	1	403	
7	102	92	20	1	403	
8	102	98	20	2	403	
9	102	96	20	2	223	
10	102	92	20	1	393	
11	102	92	20	4	1143	
12	102	98	20	2	263	
13	102	98	22	1	343	
14	102	98	22	3	403	
15	102	84	22	2	403	
16	102	84	22	1	363	
17	102	84	22	2	503	
18	102	84	22	2		
19	102	86	20	1		
20	102	86	20	1		
21	102	86	20	1		
22	102	86	20	1		
23	102	86	20	1		
24	102	86	20	1		

DATE	TEMP.	PULSE	RESPIRATIONS	MOTIONS	URINE	REMARKS
1914 25	102	120	21	2	?	Admitted
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27	102	100	18	1	157	
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1	102	106	18	2	213	
2	102	100	18	3	193	
3	102	120	20	2	243	
4	102	96	16	2	333	
5	102	90	16	2	333	
6	102	92	20	1	403	
7	102	92	20	1	403	
8	102	98	20	2	403	
9	102	96	20	2	223	
10	102	92	20	1	393	
11	102	92	20	4	1143	
12	102	98	20	2	263	
13	102	98	22	1	343	
14	102	98	22	3	403	
15	102	84	22	2	403	
16	102	84	22	1	363	
17	102	84	22	2	503	
18	102	84	22	2		
19	102	86	20	1		
20	102	86	20	1		
21	102	86	20	1		
22	102	86	20	1		
23	102	86	20	1		
24	102	86	20	1		

WHOLESALE & EXPORT DRUGGISTS, SURGICAL INSTRUMENT MAKERS & c. BRISTOL.

